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PARALYSIS AGITANS AND SARCOMA.

REPORT OF A CASE, WITH AUTOPSY.

By Charles L. Dana, M.D.,
of New York.

My interest in the pathology and pathological anatomy of paralysis agitans began some six years ago, when I read a paper before the Philadelphia Neurological Society upon this subject, and reported the results of an examination of two cases, with autopsies.[1]

A third case was reported by me in the *Post-Graduate* for July, 1896, special neurological number, page 332.

I recently, with the assistance of Dr. Fraenkel and Dr. Muskins, have been able to make a study of the pathological findings in a fourth case, and it is the result of this which I wish to submit. The patient was an inmate of the Montefiore Home, and most of the clinical study was made by Dr. Fraenkel, to whose notes I am indebted. The pathological examination was made by myself and by Dr. L. J. J. Muskins, who contributes some specially careful and laborious studies as to the degree of degenerative change in the cells.

The history of the case is as follows:

J. B., aged sixty-one years, occupation peddler, nativity Hungary, resident of the United States seventeen years. The family history shows us nothing of importance; father dying at sixty-seven, neither parent having any nervous or mental disease. The patient married at twenty-one, and had nine living and several still-born children; had malaria, but denied syphilis, and had been moderate in the use of alcohol and tobacco. Present disease began in 1891, seven years before

[1] Philadelphia Neurological Society, February 27, 1893; New York Medical Journal, June 10, 1893.

his death. The cause is unknown, but the patient ascribed it to a fright and fall on the street. The first symptoms were tremor in the left hand, followed by some weakness in that arm. The tremor progressed its usual way, from arm to leg, and from the left side to the right. It involved also, rather early, his speech. He was admitted to the Montefiore Home in 1897. At that time the examination by Dr. Fraenkel showed the following condition: His mental condition is fairly good, though he is slow in thought and not always correct in his calculations. His memory is good. Emotional condition is rather active, as is usually the case in this disease. The speech is monotonous, slow, and at times unintelligible. His physiognomy presents the flushed face and mask-like appearance characteristic of Parkinson's disease. He has the tremor in the arms and head, also in the legs to a less extent. This tremor, while in general slight and stable, increases at times on intentional movements. There is a very considerable rigidity of the limbs, as well as weakness, so that the patient walks with difficulty. He has also the usual trouble in initiating movement; thus when he first begins to rise from a chair he fails, and has to make several consecutive efforts before he finally succeeds. After about twelve or fifteen attempts he gets up quickly, uttering a sigh of relief; all the time his lips, hands, feet, and legs are trembling with more or less intensity. His station is that of the pronounced paralysis agitans type, and the gait is equally characteristic, the steps being quick and short, with a tendency to retropulsion when the patient stops.

The tremor affects the lips, the neck muscles, the arms and hands, and the legs equally. The head is flexed, trunk flexed, upper extremities adducted at the shoulder-joint, flexed at elbow-joint and the metacarpo-phalangeal joints, and extended in all the phalangeal joints. There is flexion at the knees and hips. The patient looks emaciated and very senile. The skin is dry and scabby, and in parts dotted with small hyperæmic-looking spots all over the body, showing the most of these disturbances in small or large areas, and localized, warty-like growths in the lower extremities, especially on the left. Some of these warts are ulcerating and discharging a bad-smelling hemorrhagic fluid.

The tongue is coated thickly, and when protruded trembles very perceptibly. Pharyngeal reflex is present and motility of soft palate undisturbed. There is inability to wrinkle the forehead, but the motility of the lower facial branches is normal. Pupils small, reaction good. Ocular movements very slow, and the patient cannot reach the extreme lateral positions. Vision and hearing seem normal, as do also smell and taste. There is some stiffness of the jaw and difficulty in swallowing. There is no disturbance of the sensibility of the skin. There is no ataxia or inco-ordination. Superficial reflexes are present and moderately lively; knee-jerks are present.

On electrical examination and stimulation there is a decided quantitative diminution of the direct reaction, and contraction is vermiform.

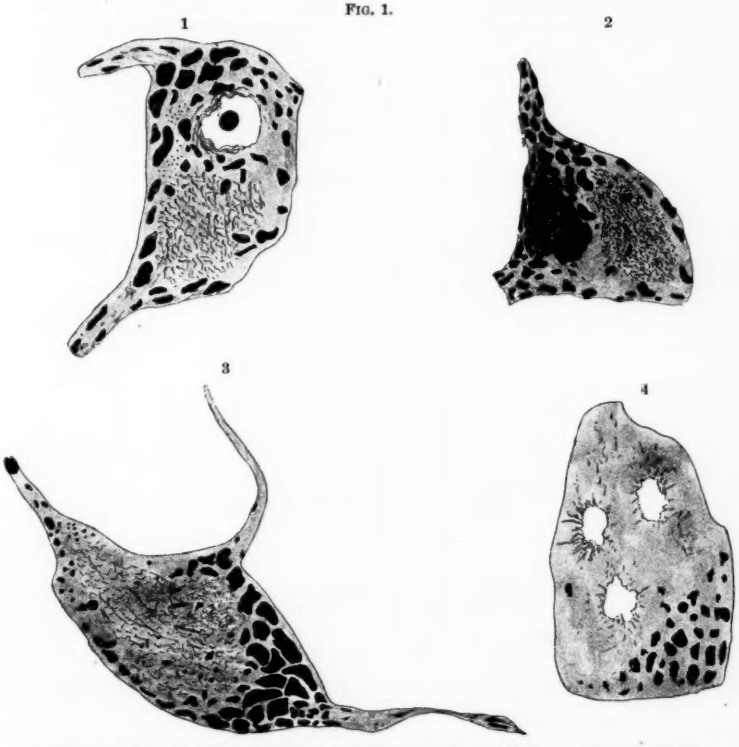
The condition outside of the nervous and muscular field is as follows: The pulse is 72, somewhat hard; temperature, 97.5°; respiration normal; heart and lungs are normal. Manner normal, and there are no anatomical stigmata of degeneration. The mouth is without teeth.

The patient's condition did not markedly change during the rest of his life, except that there was a steady increase in weakness, rigidity, and emaciation. The warty and ulcerating nodules of the skin, which

were diagnosed as sarcoma, increased and added to the exhaustion of the patient, who died finally of exhaustion after his illness had lasted *eight years*.

Post-mortem examination was made by Dr. Onuf, who carefully preserved the cord, brain, and parts of the sympathetic in formalin, alcohol, and in Mueller's fluid. The post-mortem, outside of the nervous centres, showed no lesion of importance except the cutaneous sarcoma.

FIG. 1.



1. Anterior horn cell, second lumbar, showing central pigmentation. 2. Second lumbar, lateral displacement of nucleus. 3. Anterior horn cell, upper dorsal, central pigmentation. 4. First dorsal, vacuolation loss of nucleus. (Nissl stain. 1/12 objective.) (MUSKINS.)

Examination of the Spinal Cord. Nissl stains were made by Dr. Muskins of the sections taken from the mid-cervical, upper dorsal, third and fourth dorsal, seventh dorsal, middle lumbar, and upper sacral regions. These showed very striking degenerative changes in the cells of the anterior horns. The changes consisted in marked pigmentation of the cell-body, chromatolyses, loss and displacement of the nucleus, atrophy, and loss of some of the dendrites. The absence of or defect in dendrites is common (Fig. 1). The pigmentation occupied, in some

cases, the periphery, involving nearly half the body of the cell, in other cases attacking the centre of the cell-body and following it out, leaving a peripheral portion of fairly healthy substance. In many cases the pigmented part had apparently dropped out, leaving vacuoles, and vacuolization of the cells is a more striking phenomenon than is often seen. This, I think, however, may be largely due to the bad preservation of the specimen, or, at least, due to a post-mortem chemical and mechanical change, caused by putting the tissue through various hardening processes.

An attempt was made, at my suggestion, by Dr. Muskins to estimate the percentage of degenerative cells at different levels. The cells were

FIG. 2.

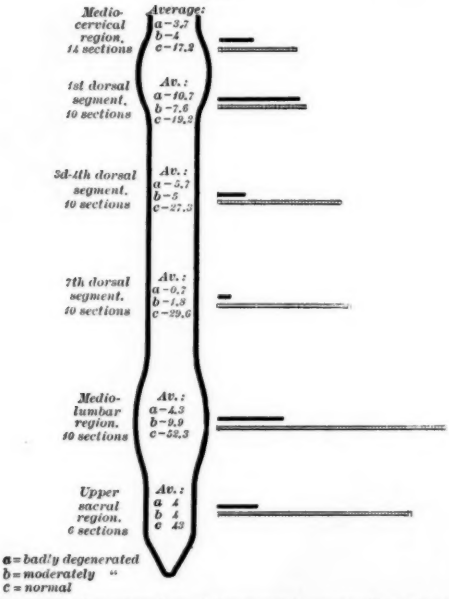


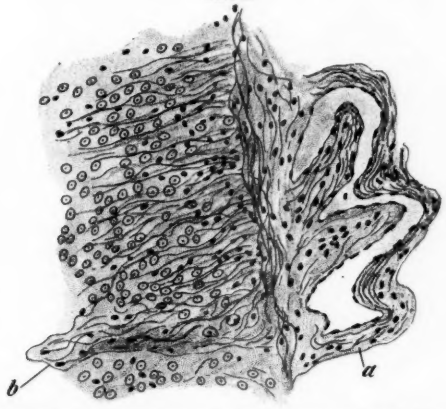
Diagram showing the relative percentage of degenerated nerve-cells in the anterior horn. Dark lines represent cells *a* and *b*.

divided into three categories: (*a*) Those that were extensively pigmented and degenerated, (*b*) those moderately so, and (*c*) those that were nearly normal. Cells of these three categories were counted in the different levels and percentages made out. In all the cells in sixty sections at different levels were counted. The percentages of cells indicated in group *c*, and degenerated cells, indicated by groups *a* and *b*, are shown diagrammatically in light and dark lines, respectively, upon the accompanying diagram (Fig. 2). It will be seen that the greatest proportion of degenerated cells was in the lower cervical and first dorsal sections—the area where the tremor first starts; next to that in the mid-cervical region, then in the mid-lumbar and upper sacral regions. The least affected portion seemed to be the middle dorsal and upper dorsal.

Examination of the columns of Clarke showed some degenerative changes, but, on the whole, their condition was fairly good, certainly much better proportionately than that of the cells of the anterior horns. It is impossible to place any very great weight upon the changes in this area.

The study of sections stained by carmine and Delafield stains, for purposes of determining the condition of connective tissue, showed slight increase in the interstitial connective tissue of the white matter. This is more marked in the marginal columns than in either the pyramidal tracts or the posterior columns, but it is not a very striking increase in either case. The anterior roots showed a greater amount of connective tissue thickening than the posterior. In neither case were the roots very much affected. Stains by Weigert-Pal method showed no very marked change.

FIG. 3.



Showing tortuous vein in posterior septum and slight connective tissue infiltration in posterior column. *a*, Vessel. *b*, Connective tissue. (Delafield stain.) (MUSKINS.)

Stains by the Marchi method were negative, except to show very strikingly degeneration of the cornual cells. The degree of degeneration was shown to be more extensive than was revealed by the Nissl stain. *There was no reaction in the white matter.*

Throughout the whole area of the cord, both in the gray and white matter, there were many large vacuolar areas, such as are often seen in degenerated spinal cords that have been hardened in alcohol. They indicate not only that there has been some general atrophy of the cord and connective tissue proliferation, but probably also some corresponding vascular congestion and dilatation of perivascular areas (Fig. 3).

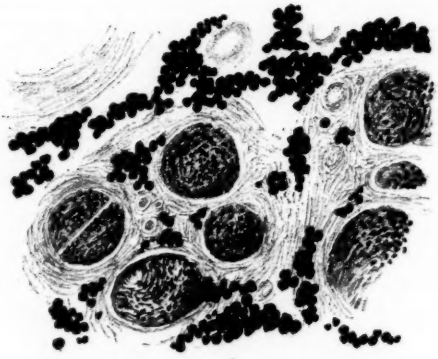
Occasionally one could see thickened bloodvessels, but on the whole the changes were slight. *There was no general arterial sclerosis.*

The sciatic and ulnar nerves were perfectly normal to Marchi and Weigert stains (Fig. 4).

Brain. The cells of the central convolutions show a considerable degree of degeneration. They are not much pigmented, but the nuclei

are often very large, and are surrounded by a bright area, showing that they are splitting away from the body; they are sometimes seen lying at the edge of the cell. There is chromatolysis, making the cell-body look like an open network—a kind of granular degeneration seen also in a previous case described by me. The cells are not much atrophied,

FIG. 4.



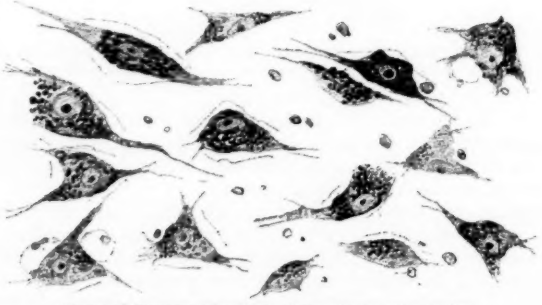
Ulnar nerve.

and the processes look fairly well. There is not much vascular or neurogliar change. Only the central convolutions were examined.

The *cerebellum* was normal.

The *medulla* showed some degeneration of motor nuclei cells—not as marked as in the cord.

FIG. 5.



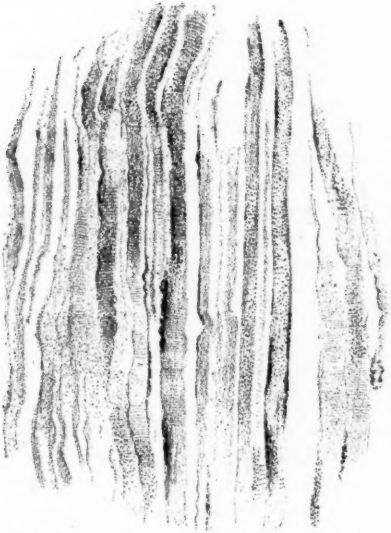
Group of degenerated cells of olivary body. ¼ objective.

The *inferior olivary body-cells* showed moderate degeneration in nearly all cells, a change that has been noted by others (Fig. 5).

Examination of the Muscles. A piece of muscle from the adductor pollicis and another from the palmaris brevis were removed and stained with picro-carmine, and afterward by the Marchi method. The picric acid stain showed a striking difference between the coloring of different

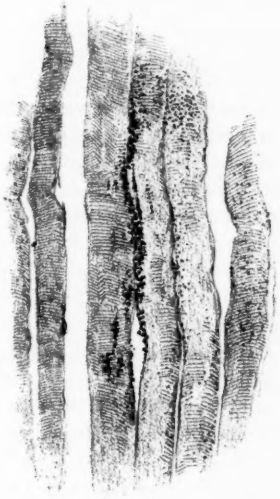
muscle fibres, which was thought to be due, perhaps, to accidental differences in the impregnation with the dye. Examination by the Marchi method, however, showed that a large number of the fibres took on a dark stain, and were full of minute black granules of osmic acid, due to the presence of fat. Upon closer examination it could be seen that there was a slight fatty degeneration of many of the muscular fibres, while the others were apparently perfectly healthy. This staining was usually rather diffuse, but here and there in nearly every field one could see little patches of particularly dark staining, which appeared to be the nerve terminations (end-plates) upon the muscle. Staining by hæmatoxylin showed no increase in the nuclei, no proliferation of connective tissue, and no disease of the bloodvessels. The muscle fibres, apart from

FIG. 6.



Fatty degeneration of muscle (adductor brevis). ⅔ objective.

FIG. 7.



Fatty degeneration of muscle (adductor brevis). ⅙ objective.

the peculiar parenchymatous degeneration shown by the Marchi stain, were healthy in every way, there being no particular swelling nor breaking up of the fibres anywhere, and the striæ were distinct (Figs. 6 and 7). This degeneration of muscle fibre and of end-plates in paralysis agitans is, so far as I am aware, a new observation in the pathological anatomy of the disease, and I must credit my assistant, Dr. Muskins, with preparing the specimen and calling my attention to it.[1]

[1] Under the head of "Intrafibrillary Fatty Degeneration of the Muscle Fibres in a Hemiatrophic Tongue in Tabes," Prof. H. Obersteiner describes (Arbeiten, 1895, iii. p. 183) a somewhat similar condition. In this case there was no nuclear lesion, and the degeneration was ascribed to neuritis. In my case, as the drawing shows, section of the ulnar nerve showed perfectly normal fibres, and my case also the degeneration shows itself more localized, as though end-plates were affected.

The recent observations upon the Marchi stain, particularly by Nonne, seem to show that in its light degrees it indicates *trophic* disturbance of the nerve-tissues, and probably of muscle tissues, and that this degeneration is not incompatible with the *functional integrity* of the organ. It is interesting to find that a muscle which has undergone as many contractions in the last eight years as had this particular one shows this peculiar degeneration alone. The tremor in my patient's case early affected the hand muscles, and, estimating that the tremor was continuous, as in fact it was, from the beginning, during the waking hours, and that the contractions occurred at the rate of about five per second, during sixteen waking hours for eight years, I estimate that this particular bit of muscle had contracted during that time about *eight hundred and forty million times.*

SUMMARY. The anatomical changes may be summed up, then, as follows: Some general atrophy of the cord, moderate increase in connective tissue, and dilatation of perivascular spaces; no special thickening of the bloodvessels, either veins or arteries; no meningeal thickening; pronounced changes in the anterior cornual cells, consisting of atrophy of these cells, pigmentation and vacuolization, loss of dendrites, diminution in the richness of the plexus formed by the dendritic processes; the process most marked in the upper cervical and lower dorsal region; Clarke's column is not markedly affected; the brain cortex shows slight degeneration of cells; the olivary nerve-cells degenerated; the peripheral nerves normal; the muscles and end-plates showing some fatty degeneration.

COMMENTS ON THE PATHOLOGY AND PATHOGENY OF THE DISEASE. There are two views prevailing regarding the pathological anatomy of paralysis agitans. One is that it represents merely a premature condition of senility, and that the changes are the same as those found in extreme old age, the differences being a matter of degree, not of kind.[1] The other view is that the nervous system in cases of paralysis agitans presents changes which differ from those of senility, and although these have not yet been thoroughly studied

[1] In a recent article by Nonne upon the spinal cord changes in cases of pernicious anæmia (Deutsche Zeitschrift für Nervenheilkunde, vol. xiv, Parts III and IV, p. 192), the author states that he has examined the spinal cords of ten old people, and also the spinal cords in two cases of paralysis agitans, and that he does not find any differences between the conditions in these two cases, nor could he tell the difference in the appearances between the cord of a very old person and that of a person who had had paralysis agitans. To this I can only say that, in the first place, Nonne gives no account of how he has examined the cords of his cases of paralysis agitans, nor whether he has used all possible means of examining them by Marchi's method, the Nissl stain, and so on. In the second place, I can show from my specimens that the cord in the case of paralysis agitans here studied by me does not present the appearance of the senile cord as described by Nonne. There is not the extreme sclerosis of neuroglia increase, and the bloodvessels are positively very little diseased. Third, I must say, again, that paralysis agitans is clinically entirely different from old age, and that if we have not yet discovered the pathological anatomy, it does not follow that we shall not do so, or that the question is fully settled.

out, further research will in time discover them. It is this latter view which I most decidedly share. There are, to be sure, many changes in the spinal cord, brain, and even nervous system of paralysis agitans which are more or less identical with those found in the senile nervous system. But to say that paralysis agitans has underlying it nothing but the premature senility is to go utterly against the facts of clinical experience. The symptoms of rigidity and palsy are not those of premature senility. The vasomotor disturbances, the burning and lightning pains, the peculiar rigidity, the hitching of the voice, the sharp interruptions in volitional efforts, are also unlike the phenomena of senility. Even the tremor is essentially different, for the tremor of old age is larger in sweep and involves the head more often, and interferes relatively less with the active movements and comfort of the individual. The tremor of old age is benign as compared with the crippling tremulousness of Parkinson's disease. There is, indeed, little room for argument upon this point. The dissimilarity is so striking, and the symptoms in paralysis agitans are so progressive, severe, and malignant, that it is impossible to believe that there is not a distinctly different anatomical change underlying them. There is, probably, a common condition of increase in interstitial tissue and of proliferative neuroglia natural to advanced years; but I do not find in my cases of paralysis agitans such profound changes in the bloodvessels or such extensive arterial sclerosis as is seen in extreme old age. The pigmentation of the cornual cells exists in both cases, but it may yet be discovered that the neuronic degenerations in paralysis agitans are peculiar in degree and localization, as well as in the way in which they involve the cell-bodies.

The history of the disease shows that it is primarily a parenchymatous nervous disease. The onset of the trouble is sometimes very sudden, and shows the almost apoplectic-like disturbances of certain groups of nerve-cells. Where and what is this disturbance?

The clinical facts and the pathological examinations in cases of paralysis agitans lead one fairly to infer that the incidence of the attack falls (1) upon the dendrites of the cells of the anterior horns of the spinal cord or (2) else upon the end-brushes of the neuraxons that come from the voluntary motor tract and the cortical motor centres of the brain. We might in the latter case establish even a sort of analogy between paralysis agitans and locomotor ataxia. Thus while in locomotor ataxia the effect of the pathogenic poison is to produce a degeneration of the axis-cylinder of the posterior spinal ganglion cells, this degeneration, often apparently beginning in the periphery, and not closely involving the cell-body at first, in paralysis agitans the special poison affects the end-brushes of the axis-cylinders of the cortical cells of the brain, producing a destruction of them, but not involving the main trunk of the axis-cylinder.

This view is somewhat contraindicated by the fact that even in very old cases of paralysis agitans there is no special involvement of the pyramidal tracts.[1] On the other hand, in the very severe cases there is a correspondingly greater destruction of the anterior horn cells, and this destruction especially involves the dendrites and the cell-body, while, even in the bad cases, one sees the axis-cylinder process and the anterior roots still in comparatively normal condition. The most logical conclusion, therefore, that one can reach is that in paralysis agitans there is early a functional disturbance and later a degeneration and destruction of the dendrites of the anterior horn-cells which interfere with the even flow of motor impulses, and, finally, lead to motor weakness and rigidity, owing to the cell being practically cut off from the brain. The fact that in paralysis agitans there is rigidity, but no great increase in the deep or superficial reflexes, is explained also by this interference in the functional activity of the dendrites and the easy flow of impulses from cortical to spinal neuron, and from sensory to motor neuron; for, on account of this, the completeness of the spinal reflex arc is impaired. The difference between this condition and that found in spastic paraplegia, due to a sclerosis of the voluntary motor tracts, is manifest, for there the dendrites of the anterior horns which subserve reflex purposes are normal, while in paralysis agitans all are somewhat affected. The rigidity of this disease is much like that found late after total transverse cord lesions.

PATHOGENY. It will, I think, be found that there is quite a number of degenerative diseases of the nervous system which are due to remote or secondary results of infections and poisons. Syphilis, for example, produces two results: one a direct injury by its exudates and irritating poison, the other a later, slowly degenerative process, due to some paralyzing effect which has been put upon the cell-life. It seems as though the circulation in the body of the luetic poison lessened the longevity of certain groups of nerve-cells, so that after ten or fifteen years the poisoned nerve-cell begins to die. In the same way I have seen persons subjected for years to slight degrees of lead-poisoning suffer at an early stage from a temporary lead paralysis, recover completely, then, twenty or thirty years later, undergo a progressive degenerative disease involving the peripheral nerves and the spinal cord, ending in an extensive sclerosis and terminal softening. The acute infection which causes anterior poliomyelitis is usually cured, and apparently the poison eliminated from the system; but sometimes twenty years after a degenerative process sets up in the anterior cornual cells, which slowly progresses until they all die, one after the other, and the patient himself becomes a paralytic wreck from progressive muscular atrophy.

[1] Marchi stains do not at all affect the white columns of the cord, but show strongly the cellular degeneration.

I have not space to expand this view of the subject of the secondary results of infection, but I believe it may be developed into the dignity of a broad pathological law, and this, I think, will in time give us some clue to the pathogeny of paralysis agitans. Sometime in the career of these cases the patients have been subjected to an infection or poison from which they have recovered. It has, however, left the imprint upon certain groups of cells and neuronic mechanisms, and when the degenerative period of life comes these less viable structures begin to go to pieces. What the primary infection in paralysis agitans is I do not know, but perhaps some autochthonous poison allied to that of rheumatism. At any rate, there is no other preceding history that is so often obtainable as one of rheumatism, and there are many facts in the clinical history of the disease which suggest a relationship between paralysis agitans and that form of rheumatic manifestation which is known as rheumatoid arthritis.[1]

A STUDY OF A CASE OF FAMILY PERIODIC PARALYSIS.[2]

By John K. Mitchell, M.D.,
of Philadelphia.

The curious condition of periodic paralysis is one of the utmost rarity, and in this country but three cases have been seen, with a doubtful fourth, previous to the ones herein reported. I should omit from the consideration the cases of Rich, where the paralysis was directly due to a hereditary susceptibility to cold, and was distinctly spasmodic in character. Gibney's cases, supposed by him malarial in origin, certainly do not belong to this group either.

I do not propose to enter into the history of the previous study of this disorder except incidentally. An admirable paper by Dr. E. W. Taylor, in the *Journal of Nervous and Mental Disease* for September and October, 1898, contains a very complete bibliography of the previous work, with analyses of all the cases hitherto reported, and to this I am not able to add anything except my own cases.

All the cases observed have presented, with small variations, the same type—a paralysis of the voluntary muscles, usually total except for the

[1] It has long since occurred to me that possibly the disease might be the result of some defect in glandular secretion. In order to test this as far as was possible, I have treated patients with all the known original substances of the body—namely, thyroid gland, thymus, pituitary body, cerebrin, testin, adrenal, etc. These substances have been given in doses running up as high as 60 grains a day, with the exception of the thyroid extract. The results were essentially negative so far as throwing any light upon this mode of pathogenesis of the disease was concerned. The use of thyroid gland makes the patient decidedly worse. The use of the pituitary gland in large doses quieted the patient for a time, but this is only temporary, and the quieting effect I have seen produced in other cases. The other substances had no effect whatever.

[2] Read before the Association of American Physicians, May, 1899.

head and face muscles, with loss of reflexes, with absence of electrical reaction, with no mental or sensory disturbance, and occurring periodically at somewhat regular intervals. Nearly all of the cases have instances in the parents or ancestors of the patient. In the family of my patient the first one known to have suffered from it was the maternal grandfather of the present victim, a Pennsylvanian by birth, a tinsmith by trade, who had in early life seizures like those from which my patient has suffered, but much less severe. A first cousin of this same grandfather had periodic attacks of paralysis quite as bad as any which have been described. His business kept him travelling much through the rough mountain districts of central Pennsylvania, and he used to say that he was often obliged to get off his horse and "have a paralysis." Both these persons, so far as known to their descendants, ceased to have the attacks in middle life.

The mother of the patient is one of six children. Of three elder sisters none have ever had any spells of the palsy. An older brother had one attack, brought on by overexertion, but has never had another. A younger brother has suffered in the same way, but the attacks did not begin with him until he was a grown man, and have since occurred only at long intervals. A daughter of this brother, now between twelve and thirteen years of age, has periodic palsies of the same sort, and full details of these two cases follow my patient's history. None of the other cousins except this one has ever had the family affliction. The mother of the patient began to have periodic paralyses when fourteen years of age, probably at about the time when her menstruation appeared. Their approach was sometimes announced by a slight sense of helplessness, which would last for a whole day. Going to bed with this, she would wake in the morning with total muscular paralysis, usually lasting thirty-six hours, seldom longer, and never more than forty-eight. The paroxysms recurred at long and rather irregular intervals, often months apart, and after she reached adult life decreased in frequency with the advance of years, and ceased altogether at about the thirty-eighth year. She is now forty-four years of age, and presents some interesting symptoms.

Mrs. E. J. is a well-looking woman, scarcely appearing as old as she is, calls herself perfectly well, menstruates regularly, and is active mentally and physically. She has a little difficulty in walking unless the ground is perfectly smooth; a very slight inequality makes her stumble, and if she stumbles she is very apt to fall. This is especially marked at night, when she cannot see where she is going, and has been noticed for some years. When sitting she gets up with some difficulty, rising as if her legs were weak, with a sort of wiggle, and aiding herself with the nearest object upon which she can lean. She thinks this is rather worse of late years, but she had not thought it bad enough to mention

until her attention was called to it by questions. Her station is perfectly good, her pupillary reactions normal; the knee-jerk decidedly slow and small, a little better on the left than on the right; on the right there is no ankle-jerk, on the left the ankle-jerk is present. There is muscle-jerk in both arms and in the left leg, but none in the right leg. Galvanic and faradic reactions are normal, and there is no alteration in sensibility. The state of things presents a somewhat mixed character, which were it not for its very long duration, the absence of eye-symptoms and of pains one might incline to think preataxic; but if it were of tabetic nature it would be symmetrical and would not present entirely different qualities of knee-jerk upon the two sides, as it does, in these respects resembling rather the ataxic paraplegia of Gowers than true tabes. Her gait shows no abnormality whatever, but I incline to the belief that the repeated attacks of her earlier life have in some way definitely altered the lower segments of the spinal cord and impaired the control of the legs. There is no trouble with the functions of the bladder, rectum, or uterus.

The present patient, C. H. J., nineteen years of age, is a mechanic in a bicycle factory. But for the neurosis under consideration no hereditary disease is known on either side of his family. His mother's history has been given; his father is living and in excellent health; his younger brothers and sisters are well, although it may be said they have not yet reached the age at which the palsies usually appear. With the exception of the ordinary diseases of childhood his own health has been perfect, his habits good, and his life an active and wholesome one until his present trouble began, in his thirteenth year.

The onset was sudden, without known cause. He awoke one morning and found that he could not move hand or foot; he was able to turn his head from side to side, but could not raise it from the pillow. There was no pain, no loss of sensation, no impairment of function of any kind. About two months after the first attack another occurred, and for three or four years they returned at intervals of about two months, with a duration generally of forty-eight hours, nearly always coming on during the night, so that the patient went to bed feeling no more than a trifling degree of enfeeblement in the legs, and awoke in the morning quite helpless. No possible cause can be assigned in his habits or mode of life. He cannot trace any single attack to excessive exercise, emotion, or indulgence of any description, and except that when they pass he finds himself a little sore and stiff for a few hours; he cannot see that he is otherwise than perfectly well in the intervals.

The intensity of the attack has remained about the same up to the present, but for nearly a year past the frequency of occurrence has become rather greater, and for six months before he was first seen, in

October, 1898, the paralyses had recurred with remarkable regularity upon Wednesday of every week.

It is well to say at once that nothing about the patient between or during the attacks suggests the slightest possibility of a hysterical element. He is a normal, healthy boy, well developed, in admirable physicial condition, and much disgusted with the enforced idleness of this disablement. Many attacks have been seen in the hospital, and the various seizures differ only in the degree of immobility produced. One may be described in detail and stand for a type. Sometimes for a few hours in the afternoon or evening the patient notices slight weakness in the lower limbs, and if examined at this time at least one decided symptom is present—a lessening of the knee-jerk. This happened to be the case when I showed him one evening at the Philadelphia Neurological Society, when he said, after coming into the room: "I am just beginning to feel an attack coming on," and upon examination the knee-jerks were found to be capricious, diminished, and easily exhausted. This prodromal period is not always present. He may go to bed perfectly well and awake in the morning wholly paralyzed from the neck down. I say "from the neck down," but the neck to a certain extent shares in the difficulty. He can, when lying still, turn the head from side to side, but he cannot raise it from the pillow, and if placed in an erect position the head falls forward on the chest, or backward almost between the shoulders, without the possibility of any control on the patient's part.

Breathing in the attacks is somewhat shallow, and appears almost altogether diaphragmatic in character. If he is requested to cough he is unable to, or even to take a normally full inspiration. If the nares are irritated he cannot sneeze, though he feels the annoyance and has a desire to do so.

He does not have much appetite, and says he perceives slight difficulty in swallowing, but this is not evident to the observer. While the paralysis lasts he usually passes water only once or twice in the twenty-four hours and has no fecal movement. There is no disturbance of the mind; speech is not affected, and he has no pain except the discomfort of lying absolutely in one position, which somewhat annoys him. So far for his own statement of his feelings and symptoms.

Physical Examination During an Attack. The patient is notably paler in the attack than during healthy intervals. His lips are less well colored; his tongue is somewhat coated and his breath has a heavy smell, but there is no change of expression and certainly no mental impairment. He answers readily to all questions and takes an interest in the examination of his symptoms.

Upon study of the chest the breath-sounds are found to show slight general lessening, due, probably, to imperfect dilatation of the chest. On being asked to take a long breath he makes an effort to do it, but

has little control of the accessory muscles, and moves the cage of the ribs but slightly. The heart's action and pulse are decidedly changed from normal. The apex-beat is below the fifth rib, rather indistinct, two and a quarter inches from the mid-sternal line. The heart's action varies much in different attacks; it is sometimes a little irregular in time, though no distinct intermittency has been observed at any time. The heart conditions, however, were not always the same during the attacks. Examined several times during October and November in attacks, the heart presented usually a split second sound, very distinct in the pulmonary area, a systolic murmur well heard upon full expiration and limited to the same area, transmitted somewhat up and down the left side of the sternum, strong and decided, but not loud or harsh.

In the latter part of November, examining him one day with Dr. Morris J. Lewis, we both agreed that there was a soft, low, systolic murmur, best heard at full expiration at the second right interspace and very faintly upon the left side. This was a condition which was never found again. Three days later, during an interval, the split second sound was heard at the second interspace, diminishing toward the apex, with a murmur much harsher than before and of greater intensity. The most careful examination with stethoscope and phonendoscope fails to reveal any increase in the area covered by the heart dulness. The pulse is somewhat weak and small, from 80 to 90 during an attack; sometimes, with about the same speed, its tension is increased and its character distinctly dicrotic, as shown in sphygmographic tracings made repeatedly during the attacks.

The heart examinations were made over and over again. I made not less than a hundred careful examinations of the heart in the attacks, and as he was an object of interest to the other members of the hospital staff, the examinations were made many times over by them, and the general opinion confirmed the above statements, which indicate rather a relaxed condition of the heart muscle, as well as of the whole arterial system, than an acute cardiac dilatation. In this belief I am supported by the high authority of Professor J. M. Da Costa, who studied the heart during an attack.

The reflexes in the more severe paroxysms are totally lost except the reaction of the pupil. No contraction can be observed in the pectoral muscles on striking them; the abdominal recti do not contract to ordinary stimuli, the cremasteric reflex is abolished, there are no knee-jerks, and although the patient resents the attempt to secure the plantar reflex by irritation of the sole of the foot, no muscular contraction takes place. Sensation is perfect. The electrical examinaton was many times repeated during the attacks and in the intervals. In the typical attack there was practically total abolition of reaction to any form of electrical stimulation.

In milder attacks some variations upon these general symptoms were

noted. Muscle-jerks were sometimes present, the knee-jerks were occasionally found present by reinforcement, the cremasteric reflex was at times not altogether absent, though usually weakened. In the same way the degree of muscular enfeeblement varied a little. In the early part of even severe attacks, during the morning hours, the patient would be able to move his hands, perhaps his forearms, or even his toes. As the day went on the paralysis would become deeper and all movement would be abolished. Return of normal conditions followed much the same order that characterzied the onset. The knee-jerks improved a little, other reflexes reappeared, slight contractility in the muscles was discoverable, and in the course of an hour or so from the beginning of betterment the patient was almost perfectly well. Several times periods of apparent partial recovery were followed by relapses into total paralysis. Usually progress was steady and rapid, and several times we have seen a man who at six o'clock was, so far as power to move or to respond to electrical stimulation could tell, as dead as a twelve-hours' corpse, able to walk about the ward at seven o'clock with all his muscular power, his reactions and reflexes, in normal condition.

Once at least in the course of the several months the patient was under observation he seemed to have a kind of abortive attack, in which during some days he felt dull and had the same sense of lack of complete muscular power which usually precedes a paroxysm. At the same time his color was poor and his appetite not so good as usual. Knee-jerks during the whole of this period were found to be occasionally absent, though reinforcible, and at other times capriciously present, but always diminished. It should be added that this was during a time when he was taking bromide in moderate doses. One important fact developed somewhat late in the case, or at least had not been previously observed. Upon my remarking that I should give him injections of Brown-Séquard's elixir, and that it might have an effect in stimulating the sexual functions, he laughed and begged not to have it, because he said there was sufficient stimulation in the attacks already, and that very often he suffered with partial or complete erection during the period of paralysis.

[Copy of letter from the patient's maternal uncle, Mr. C. H. W., dated January 29, 1899.]

DEAR SIR: I am perfectly willing to give you all the information in my power, but hardly know where to begin. I have been subject to the attacks you refer to from the time I was twelve years old, but have never been completely prostrated since my marriage, sixteen years ago.

The disease (or spells, as we call them) would sometimes come on me during the night, and, while feeling all right at bedtime, would be perfectly helpless on waking in the morning. At other times they would come on me gradually, and I could feel myself slowly losing control of my limbs, and finally became helpless; sometimes I could, by keeping

in motion, keep from total collapse. I suffered no pain of any description unless my limbs were cramped, but was perfectly helpless, could move neither hand nor foot, but seemed to have complete control of my talking apparatus, and, with the exception of being helpless, I usually felt exceptionally well. My sense of feeling was normal, but the sense of motion completely gone. I noticed that the attacks were more frequent in damp weather; also, that they were sometimes brought on by unusual exertion.

My daughter has suffered from the same disease, but has been completely prostrated only a few times, and as it happened at a time when I was away from home, I will hand your letter to our family physician, Dr. H. L. McIlhenny.

[Copy of letter received from Dr. H. L. McIlhenny, Norwich, Kansas, March 10, 1899.]

My Dear Doctor: In reply to your letter of February 2d, will say that I am personally acquainted with Mr. W. and family, to whom you refer in your letter.

Referring to the peculiar family affliction, I have to say that I have seen Mr. W. a number of times suffering with these mysterious attacks, though he has suffered but few in the last few years. The condition is surely a most singular one, never having seen any cases like his or his daughter's, and have never seen any authority covering these cases.

Mr. W., I understand, has given you the details of his case, which are evidently very similar to the attacks with which J. suffers. As to Mr. W.'s daughter, who is now about fifteen years of age, I have seen her in this peculiar condition but once, about one year ago, although she has had two or three attacks since, but they were rather light.

At the time I saw her she had gone to bed in the evening as well as usual, and on waking in the morning she found herself completely helpless—paralyzed. I was called and found her in this paralyzed condition—could not move hand or foot. In all respects the attack was similar to those from which her father suffers, except that she suffered quite severe pains in back and head. All reflexes seemed weakened or absent. She remained in this condition ten or twelve hours, when she began to get better, and by the next day she could help herself and began to sit up. In three or four days she was as well as usual. There does not seem to be any regularity in her attacks, nor do I think that menstruation has any relation to the attacks. I hardly think mental emotion or physical exertion are exciting causes. This young lady has been away from home several months attending school, and I have not heard of her having an attack recently. I forgot to say that in this case (Mr. W.'s daughter) I did not make a *careful* examination of the heart, only to note that the heart seemed all right.

The electrical examinations are of great interest and have been often and carefully made by Dr. J. H. W. Rhein, electrician to the Infirmary for Nervous Diseases.

In the first examination during an attack it was impossible to get any reaction either with faradic or galvanic currents in most of the muscles with the largest amount of currents bearable. With from 30 to 40 milliampères slight response could be obtained in the anterior thigh muscles. It was impossible to complete the examination, on

account of the pain caused. In examining a large pad was always placed over the sacral region and a small, interrupting electrode over the motor points of the muscle tested. The K. CL. C. contracture was always greater than the AN. CL. C. contracture.

On the second day of the same attack it was possible to get contracture here and there; for instance, the anterior tibial responded with 20 milliampères, the posterior with 18 milliampères, the sciatic nerves with 20 milliampères, and the biceps of the leg with 30 milliampères. No contraction of the rectus femoris could be obtained with less than 30 milliampères. It was necessary to discontinue the examination, as the strong currents made the boy feel faint and decidedly affected his heart.

In a second attack there was no faradic reaction whatever in most of the muscles. In the erector spinæ muscles there was fair contraction with the greatest amount of current that could be applied. The sternocleidomastoid responded slightly with largest amount of faradic current. This was true also of the other cervical muscles. With the galvanic current no response could be elicited in the cervical muscles with 20 milliampères. It was thought unwise to use larger quantities of this current in the neck region,

Examination in a third attack showed that the anterior tibial muscle responded with 20 milliampères on both sides, the posterior tibial with 12 milliampères on the right side and with 40 milliampères on the left, the erector spinæ with 20 milliampères, the abdominal muscles with 16 milliampères, the biceps, triceps, extensors, flexors, and deltoid responded with currents varying from 30 to 40 milliampères.

In the fourth examination during a mild attack the following conditions were found :

	Right.		Left.	
No contracture with	15	Anterior tibial,	15	No contracture with
	10	Posterior tibial,	8	
	10	Rectus,	18	
	15	Biceps,	15	
	20	Gluteus,	20	
	20	Erector spinæ,	20	
	41½	Flexors,	7	
	6	Extensors,	15	
	10	Triceps,	10	
	10	Biceps,	8	
	12	Pectoralis,	12	
	8	Rhomboid,	8	
	7	Sternocleidomastoid,	8	
	14	Supraspinatus,	12	
	12	Infraspinatus,	7½	
	3	Facial,	4	
	15	Deltoid,	12	
	15	Abdominal rectus,	15	
	15	Lumbar,	15	
	14	Latissimus dorsi,	14	

Biceps and anterior muscles responded very slightly to the greatest amount of current obtainable in a faradic battery, while the rectus and posterior tibial muscles responded fairly well on the left side. The posterior tibial and rectus on the right side reacted better than the biceps and the anterior group on the right side. The flexors of the arms contracted much more readily than the extensors, the biceps more readily than the triceps; the deltoids responded fairly well, the pectorals much more readily, the abdominal muscles only slightly; the infraspinatus responded fairly on both sides. No movement was obtainable in the supraspinatus on either side. The rhomboid muscles contracted quite well.

Median nerve transmission was 15 milliampères right and left. In order to lessen skin resistance as a factor, examination was also made with the limb immersed in water, but this did not seem to diminish the amount of resistance offered by the muscles to the faradic current.

During a profound attack, in which it required from 20 to 30 milliampères, CA. CL. C. being greater than AN. CL. C., to develop contraction by the usual method, when a large electrode was placed upon the sacral region and a gold needle inserted deep into the body of a muscle, it was found that fair contracture could be induced with 8 milliampères, CA. CL. C. greater than the AN. CL. C., interrupting current at the needle insertion into the muscle. On repeating this experiment upon a healthy person it was found that less than half this amount of current was required to evoke contraction in his muscles.

There has never been at any time any suggestion of reversal of the poles; C. CL. C. has always been greater than AN. CL. C. in these examinations.

When the nerves were tested the same lowered irritability was observed—that is to say, if it took 20 milliampères to induce contraction in a group of muscles, no less was required to bring about contraction when the nerve was stimulated.

Upon examining the patient during the interval and a few hours after the paralysis had disappeared, it was found that the muscles responded normally to faradic and galvanic currents, and that nerve transmission was in no way interfered with. This was repeatedly confirmed.

All these are elaborately detailed, because of the interest which attaches to the very absence of all possibility of stimulation, but may be summed up by saying that during the attack, if it approached at all to completeness of loss of power, no electrical reaction was present to any current which it was possible to use upon a living human muscle, and even when the skin was pierced and electricity applied directly to the muscle a current very much greater than that which would move a muscle in normal condition was required to produce contraction.

The examination of the urine has been most painstakingly made for me by Dr. A. E. Taylor, at the Pepper Clinical Laboratory of the University of Pennsylvania, and his report is as follows:

Urine of Period of Attack, 935 *c.c.*

Total nitrogen, 13.749 g.	Total phosphates, 1.414 g. P_2O_5.
Urea, 24.510 g.	Alkaline phosphates, 0.574 g. P_2O_5.
Urea-nitrogen = 83 + per cent. of total nitrogen.	Earthy phosphates, 0.840 g. P_2O_5.
	Total sulphates, 1.356 g. SO_3.
Uric acid, 0.526 g.	Preformed sulphates, 0.140 g. SO_3.
Xanthin bases, 0.283 g.	Ethereal sulphates, 0.216 g. SO_3.

Urine of Interval, 1610 *c.c.*

Total nitrogen, 9.397 g.	Total phosphates, 1.306 g. P_2O_5.
Urea, 18.490 g.	Alkaline phosphates, 0.494, g. P_2O_5.
Urea nitrogen = 82 + per cent. of total nitrogen.	Earthy phosphates, 0.812 g. P_2O_5.
	Total sulphates, 0.695 g. SO_3.
Uric acid, 0.194 g.	Preformed sulphates, 0.546 g. SO_3.
Xanthin bases, 0.142 g.	Ethereal sulphates, 0.139 g. SO_3.

Urine of Period just following an Attack, 1725 *c.c.*

Total nitrogen, 16 332 g.	Total phosphates, 3.084 g. P_2O_5.
Urea, 31.111 g.	Alkaline phosphates, 1.999 g. P_2O_5.
Urea-nitrogen = 88 + per cent. of total nitrogen.	Earthy phosphates, 1.085 g. P_2O_5.
	Total sulphates, 2.174 g. SO_3.
Uric acid, 0.297 g.	Preformed sulphates, 1.975 g. SO_3.
Xanthin bases, 0.090 g.	Ethereal sulphates, 0.199 g. SO_3.

Urine of Period of Attack, 1550 *c.c.*

Total nitrogen, 7.247 g.	Alkaline phosphates, 1.163 g. P_2O_5.
Urea, determination spoiled.	Earthy phosphates, 0.620 g. P_2O_5.
Uric acid, 0.103 g.	Total sulphates, 0.652 g. SO_3.
Xanthin bases, 0.0214 g.	Preformed sulphates, 0.561 g. SO_3.
Total phosphates, 1.783 g. P_2O_5.	Ethereal sulphates, 0.089 g. SO_3.

Each set of analyses comprises the figures for the urine of twenty-four hours. The first two sets were done in the period when the patient was upon milk diet; the last two were done while the patient was upon a mixed diet.

The total nitrogen and the urea, as well as the ratio between the urea-nitrogen and the total nitrogen, were within normal limits, in consideration of the diet.

The quantities of uric acid were, with the exception of the first analysis, notably low. There was, however, no constant relation; the highest and lowest figures were gotten for the urine voided during attacks, while the figures for urine voided between the attacks lie between them. Such fluctuations may, however, be seen in the urine of healthy people.

The xanthin bases were in three of the four examinations excessive. In the urine of the last attack, however, the quantity was entirely normal. Such a quantity as 0.090 g. may often be seen in various diseased states, but figures such as 0.283 g. are rarely seen outside of leukæmia. Whether the quantities of uric acid were small because the xanthin bases were eliminated as formed and not oxidized to uric acid cannot be shown.

The elimination of total phosphates was within the normal limits. In the first two analyses a reversion of the normal ratio between the alkaline and the earthy phosphates was determined; in the last two analyses the ratios were normal. This reversion of the ratio between the alkaline and earthy phosphates is considered by Gilles de la Tourette to be diagnostic of hysteria. It is, however, found in very few cases of hysteria in this country.

The total sulphates were in all analyses within the normal limits. The normal figures for the preformed sulphates indicate that the proteid metabolism was normal. The quantities of ethereal sulphates were low. The diet and the colonic lavage obviously have tended to limit the formation of the ethereal sulphates, and the figures certainly indicate that there was no excess of proteid decomposition in the alimentary tract.

The urine of twenty-six days, including three attacks, was carefully tested for ptomaïnes, alkaloids, and alkaloidal substances by the Brieger and the Baumann-Udransky methods. No trace of such substances could be detected. These tests do not reveal bacterial toxines in the true sense of the term, and a rôle on their part in the case cannot be excluded.

The urotoxic coefficent (determined according to the Bouchard method, except that the injections were done under slight ether narcosis into the jugular vein) was below 0.536 for the urine voided during the attack and below 0.557 for the urine voided between the attacks—I say below, because in each case the injection was stopped at a point when the rabbits were apparently dying, but in each instance the animal recovered. In consideration of the diet, of the fact that the man was in bed, and that the injections were made into the jugular vein, the urotoxic coefficients were normal. The close approximation of the coefficients in and after the attack is striking, and speaks against regarding the seizures as corresponding to variations in the urinary toxicity. As a matter of fact, the Bouchard procedure is crude, and under all circumstances furnishes results of doubtful value. I do not believe the negative results in these injections can be taken as a serious consideration against the view that the seizures may have a toxic cause. Chemical and bacterial studies of the feces during and after several attacks gave wholly negative results, which it is not necessary to detail.

Studies of the blood under the microscope revealed little or no changes. No malarial protozoa could be found in any form. With stained slides the relative proportions and staining reactions of the different elements were found unaltered. Counts of the red and white corpuscles gave normal results, and the percentage of hæmoglobin was always high, never having been found below 90.

I asked my friend, Dr. Alfred Stengel, to repeat these examinations, which were made by myself and by my hospital assistants, that I might have the advantage of an authoritative opinion, and to undertake some experiments to determine the toxicity of the blood-serum. In bleeding the patient, to secure the necessary amount of blood for these experiments, a remarkable difficulty was experienced. When the median cephalic vein was opened during an attack the flow of blood was so slow and so small that we could hardly secure the six ounces we wanted. Thinking the opening in the vessel had not been large enough, on another occasion we cut the vein right across, but with scarcely a better result. The patient's heart-action at the time was, as usual in the paroxysms, rather rapid and feeble, with lowered pulse-tension, and on the occasion when we abstracted the full six ounces he felt decidedly light-headed before the bleeding ceased.

Dr. Stengel's report is as follows:

"*November* 22, 1898. 1. Five c.c. of serum from blood drawn during an attack injected into rabbit weighing 1550 grammes at 4.30 P.M. Was well at 6 P.M.; died during the night. Post-mortem showed abundant hemorrhages at base of right lung; less at left base. No other changes of a definite sort.

"23*d*. 2. Injected with same serum, 2½ c.c., rabbit No. 2. One hour after: Red corpuscles, 5,012,000; white, 9800. Twelve hours later: 6,000,000 red cells and 11,550 white.

"*April* 28, 1899. 3. Injected 5 c.c. of serum into rabbit weighing 2250 grammes. Rabbit alive after forty-eight hours.

"4. Injected 9 c.c. into rabbit weighing 2150 grammes. Died in thirty-six hours."

These, like the examintions of the secretions, may be said to give negative results, and certainly do not indicate any unusual degree of toxicity in the blood.

The consistency of the manifestations in the attacks is remarkable. In twenty-five attacks observed the only difference was in the degree to which power was lost, the more or less total disappearance of reflexes, and the varying occurrence of certain changes in heart-action and pulse-tension.

Almost all of the eighteen or twenty cases observed by various writers in Europe and in this country have presented symptoms very closely alike. In almost all paralysis has been periodic in its occurrence;

generally there have been instances among relatives of the sufferer; in none have any sensory changes been found, and in those in which electrical study has been made electrical changes which greatly resemble the ones here described have been present, and in the intervals of the attack the patients almost invariably give the same history of complete and perfect strength and soundness.

I say that in all the cases there have been practically no sensory changes. One described by Fischl had distinct impairment of sensibility. This case is in other ways a rather doubtful one, and might perhaps be excluded from the list.

Oppenheim and Hirsch both describe temporary slight increase of cardiac area, and both have observed murmurs upon which the diagnosis of probable mitral insufficiency was founded, a condition which lasted only during the paralysis and disappeared rapidly with the loss of power.

In one of Goldflam's cases irregularity was observed and a faint, systolic basic murmur. In mine, as has already been indicated, there was a murmur, limited to the left edge of the sternum, and best heard at the pulmonary area. This condition has varied very much in the different attacks, but this is the one which is the most common. As a rule, J.'s heart is faster in the attacks than out of them, in spite of the complete absence of muscular movement. A slight accentuation of the second sound with a tendency to splitting is present even in the intervals.

The examinations of the blood, feces, and urine have been made in most careful and detailed manner, and the results herewith call but for one comment—namely, that most minute study has revealed no fact of importance in any of them, and in the two or three cases in which these points have been thoroughly gone into, almost equally little has been learned from the examinations.

As to treatment, it may be readily supposed that this has been experimental. For several weeks during the early observations J. was not given any medicine, but led a perfectly regular and wholesome if somewhat inactive life. This was thought advisable, in view of the fact that several cases have described their paralyses as brought on by overexertion. No such cause could be assigned in the present instance, as for some weeks he lived in the hospital ward and moved about the building freely, taking the ordinary liberal house diet and being under close observation. The first theory which suggested itself, as it has to other observers, was that the trouble had an autotoxic origin. In order that there should be no retention of feces in the intestinal canal to favor this the colon was carefully washed out every second day. During this period attacks occurred exactly as regularly as they had before he came to the hospital. He was then put to bed and kept upon milk diet; the colon washing was continued, and various intestinal antiseptics were

used. The attacks recurred with about the same interval as before. He lost a little weight upon this treatment, during which also massage was used, for the sake of keeping up his circulation and of anticipating the impairment of digestion from inactivity. It was during this time that the first studies of the urine were made by Dr. Taylor, and his examination showed one of the effects of colon lavage in the absence or lessening of the ethereal sulphates. Finding the paroxysms recurring as before, he was allowed to get up and move about a little, but the amount of milk was increased, and soon after he was returned to a house diet. At this time the later examinations of the urine were made. No evidence having been secured from the urinary and fecal examinations of any toxic condition, intestinal antiseptics were stopped and an attempt was made by the use of full doses of strychnine to stimulate the spinal cord, upon the secondary assumption that this might be the source of the disturbance. Full doses of strychnine were given and rapidly increased, and if any change took place in the patient it was that the attacks appeared at rather shorter intervals than before. Strychnine had a very decided effect upon him, and it was not long before it had to be stopped. For a time he was left without any treatment, with orders to the hospital people to occupy him as much as possible and to let him go out every day for a walk. No decided difference in the frequency of paroxysms appeared under this regime, and for a time under no treatment at all the attacks, without evident cause, grew somewhat more frequent and recurred several times with only three or four days' intermission. Bromide was then given, and the first week of its administration in doses of twenty grains three times a day there was no "spell," although just previously the intervals between the attacks had been, at the utmost, four days. He was rather long in the next seizure, which was a very complete one, and when he recovered from it he had a week during which the knee-jerks were irregular—sometimes absent, sometimes slightly present—and in this whole period the patient felt dull and not so strong as usual upon his feet.

Little as we have discovered of any unusual condition of the blood, feces, or urine in this case, it seems to me impossible to attribute the attacks to any other than a toxic cause, even in face of the negative results of the chemical studies. The analogy is sufficiently striking to some other autotoxic, or partially autotoxic, disorders, in spite of the want of direct findings to support this view, and in spite of the fact that some aspects of the case are against it.

In migraine, for example, we have an instance of a disease in which family tendency is strongly evinced, which usually begins at about the age of puberty and lessens or disappears in middle life, which occurs paroxysmally and often at fairly regular intervals. The manifestations of migraine are largely cerebral, and those of the disorder we are

considering appear in the spinal centres and peripheral nerves and muscles.

As to other possibilities, it has been variously suggested that, as in the cases described by Higier, and lately in Philadelphia by Dr. J. W. McConnell, the attacks are the paralytic equivalents of epileptic fits. But in the instances where paralytic seizures have replaced epileptic paroxysms no alteration in electrical reactions have been found, so far as I know, nor any loss of reflex and direct muscular irritability, nor are heart-murmurs produced. The perfectly clear mind of the patient throughout the attack is also against this supposition, and so is the long duration of the palsy.

The suggestion of hysteria is scarcely necessary to discuss. The altered electric reactions, the absence of reflexes, and the perfectly well-balanced, wholesome moral tone of the patient at once negative this.

A poison, or more than one poison, formed at intervals or constantly formed and gradually accumulating, and acting severally upon the heart and vessels, the spinal centres, as shown by the sexual excitement, and upon peripheral nerves and muscles, is the most warrantable conclusion. It is difficult to imagine one toxic material which should have all these effects; but certainly no one could study this boy's state and not have poisoning suggested to his mind. The heavy breath, the coated tongue, the comparative suppression of urine during the attack, with an unusual flow after, all carry out the analogy to migraine. If one poison might act upon both spinal centres and peripheral nerves and muscles, it is, perhaps, not too fanciful to account for the heart conditions by supposing that the resulting total flaccid palsy may extend to the muscles of the arterial system, and that this relaxation allows of so much dilatation of the great vessels as to favor the production of a murmur.

Some additional testimony to a special action of the poison upon the cord may be drawn from the examination of the patient's mother, in whom there was distinct, if slight, enfeeblement of function in the lower spine.

On the whole, upon due consideration of all the cases that can possibly be included in periodic family paralysis, it appears to be a warrantable conclusion that two poisons are formed, one of which predominates in some cases, another in other cases; according to the predominance of one or the other, the effect is greater upon the peripheral nerves and muscles or upon the spinal centres. The disease is remarkable in this that it is clinically a most distinct entity, that all observers agree in their statements of the vast majority of symptoms, and that up to the present time the causation and pathology of the affection remain in absolute darkness.

KRAUROSIS VULVÆ:*

A DISEASE OF CHRONIC INFLAMMATORY NATURE AND CHARACTERIZED BY CUTANEOUS ATROPHY, WITH MARKED SHRINKING AND CONTRACTION OF THE VAGINAL ORIFICE.

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This exceedingly interesting and fortunately rather rare disease was first brought prominently to the attention of the medical profession by the classical article of Breisky,[4] published in 1885, in which he described at length twelve cases, and presented a clinical description of the condition which can scarcely be improved upon at the present day.

Ten years before that time, in 1875, Dr. Robert F. Weir,[1] of New York, described, under the name of ichthyosis, a condition of the vulva which, in its clinical characteristics, closely resembled kraurosis. Lawson Tait[2] seems to have noticed cases of a similar nature at about the same period that the article of Weir first appeared, which he then described as "serpiginous vascular degeneration of the nymphæ." But it was not until the observation of Breisky[4] that the disease was first looked upon as a distinct entity, apart from pruritus and the not uncommon senile atrophy of the vulva.

Clinically, the picture of this disease is most striking. The first symptoms noticed are usually those characteristic of pruritus, which consist of an intense and more or less progressive itching and burning of the vulva. In some cases the affected tissue is excessively hyperplastic and dyspareunia develops early. The skin is frequently discolored, and small red spots appear on the surface. Some time after these symptoms are noticed a peculiar shrinking of the superficial tissue of the vulva begins to take place. Discolored spots appear which are hyperæsthetic. The skin becomes dry and whitened, and often covered with a rough and thick epidermis.

The disease may be unilateral or circumscribed, but usually the tissues of the labia majora, the nymphæ, the area about the clitoris and urinary meatus all become more or less involved, while the skin about the anus is frequently affected. As the disease advances the small labia

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gradually disappear, fusing with the labia majora, and the skin becomes shiny and drawn smoothly over the shrunken clitoris, which has apparently retracted behind the skin, and is now only indicated by a small depression instead of a prominence. Underlying vessels are frequently seen through the transparent epidermis, and cracks appear on the dry surface, which extend often into the corium. A sensation of drawing and shrinking of the vulva is usually experienced, and the vaginal orifice becomes gradually narrower and more and more contracted, until frequently the little finger can scarcely be introduced, and all sexual intercourse becomes a physical impossibility. When this condition has been reached the pathological process is arrested, the subjective sensation of shrinking passes away, and the symptoms of pruritus, usually prominent only in the earlier stages of the disease, are no longer experienced. But the shrunken and contracted vaginal orifice still persists, and is never spontaneously restored.

That the disease occurs but rarely is shown by the fact that Fleischman,[5] who was especially on the watch for this condition in Breisky's clinic, in Prague, found only 8 cases in 1550 patients. On the other hand, Lewin, at the Charité, observed no case among 70,000 to 80,000 patients.

The pathological examinations of Breisky's cases, made by Fischels, form the foundation of our present knowledge. These researches showed the papillæ of the skin transformed into scar-like masses, the connective tissues of which were almost homogeneous, partly sclerotic, and without wavy fibres. The papillæ were, for the most part, low and varying in size, while the rete Malpighii was in places exceedingly thin. Sebaceous glands had disappeared altogether, and sweat glands were found to be almost entirely wanting. The microscopical examinations of Orthmann[11] in Martin's cases added still further to our knowledge of the pathology of the subject. He found some portions in a hypertrophic condition and other parts in a condition of atrophy. Under a thin horny layer the rete Malpighii was markedly narrow, some places showing but slight remains, the greater part of it having disappeared. The tissue of the corium had entirely lost its warty appearance, and was stretched and sclerotic. Here and there areas of small round-cell infiltration could be seen. The stratum Malpighii, outside the part of the true skin which was in a condition of hypertrophy, was atrophied. In other portions the horny layer was much wider, the cells being in strata of many layers upon a much-widened rete. The papillæ were much dragged out in width, and, like the corium, were more or less infiltrated with small round cells. Degenerative nerve processes were sought for, but not found, and, likewise, bacteriological examination proved negative. Up to this time, as Sänger points out, only the secondary stages of the disease had been described. The observations of Peters[16] are of especial

interest, as he described the condition of inflammatory hyperplasia of the connective tissue, with a tendency to scar-like shrinkage, and inflammation and œdema of the upper layers of the epidermis, as well as degeneration of the elastic tissue. As a result of his researches he classified the disease among the diffuse connective tissue hypertrophies of the skin. In a case studied by Neumann the horny layer of the epidermis formed ribbon-like strips of wavy course, the cell nuclei being scarcely distinguishable. Nowhere was the stratum lucidum to be seen, while the prickle cell layer was perfectly prominent.

Longyear[36] made careful microscopical examination of several cases of kraurosis, and found bands of fibrous tissue below and entirely separate from the skin and mucous membrane, in the place of, and resulting from the destruction of, the subcutaneous and submucous cellular tissue. He believes that the pathological changes begin with a fibrous tissue formation in the subcutaneous tissue, and that the pathological changes in the skin are the result of the fibroid transformation in the underlying structure; that this new-formed fibrous tissue by contraction not only puckers the mucous membrane but strangles the blood-vessels.

The pathological examination of our own case is reported as follows: The specimens consist of several narrow strips of tissue which have been removed from the muco-cutaneous surface of the vaginal orifice and hardened in 10 per cent. formalin solution. These have been mounted in celloidin, cut in transverse section, and stained with hæmatoxylin-eosin, by Van Gieson's method, acid fuchsin, carmine, and by Weigert's method for nerve-fibres. (See Figs. 3 and 4.)

A general survey of the sections under the low power shows marked lesions both of atrophy and hypertrophy in different portions of the epidermis, with a general inflammatory condition of the corium and subcutaneous tissue. Examining now carefully the epidermis in detail, we note first that the stratum corneum, stratum lucidum, stratum granulosum, and stratum Malpighii can be differentiated with great distinctness in most of the thicker portions where hypertrophy is present, while in the parts where atrophy has taken place the layers are indistinctly blended together, the stratum lucidum can scarcely be discerned, and the stratum Malpighii fades away often to a mere thread. On the free surface of the epidermis, and so closely adherent to the stratum corneum that it appears as another layer, a blood-crust is seen. This crust is made up of red blood-corpuscles, the outlines of which are distinctly apparent in most parts, with a few leucocytes caught here and there among them. At intervals along the surface cracks, or fissures in the epidermis, are found, which usually penetrate the corium, and are often filled with blood-crusts (See Figs. 5 and 6.) Near the border of healthy tissue the stratum corneum appears normal or as a narrow band of ker-

FIG. 1.

Microphotograph of normal labium majus, showing numerous hair-follicles, sebaceous glands, and bloodvessels in the subepithelial tissue.

FIG. 2.

Microphotograph of normal labium minus, showing the subepithelial tissue rich in bloodvessels, elastic and muscular fibres, but entire absence of hair-follicles and sebaceous glands.

atinous tissue, with protoplasmic outlines indistinct, and with no nuclei. As we follow it into the diseased portion it becomes enormously thick-

FIG. 3.

Microphotograph of labium, showing great hypertrophy of epidermis. (See Figs. 1 and 6. Blood-clot upon the surface, and entire absence of sebaceous glands and hair-follicles in the corium.

ened in some areas, and in other localities almost entirely disappears. No protoplasmic outline can be at all discerned, the hæmatoxylin stain

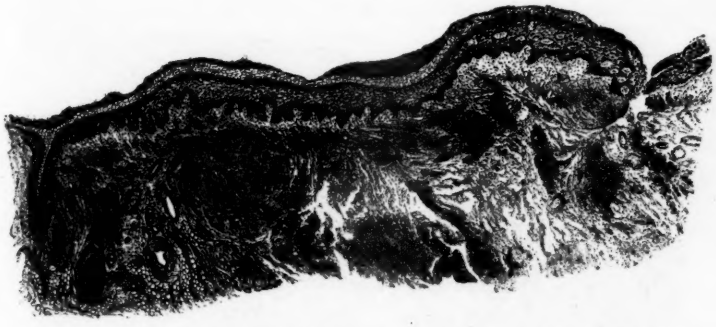
FIG. 4.

Microphotograph of labium, showing enormous hypertrophy of stratum corneum and stratum Malpighii; epithelial pearls in the epidermis.

is taken indistinctly, or not at all, and there is a marked tendency to the formation of whirls and twists resembling epithelial pearls. Free red blood-corpuscles are also occasionally present, and degenerate hair-

follicles are discoverable, both in longitudinal and cross-section, the shafts of which are degenerate and often replaced by blood-cells. Keratin is seen here in great excess, the protoplasmic outlines are almost entirely lost, and the stratum corneum appears like a homogeneous fibrous band. (See Figs. 3 and 4.) The stratum lucidum can be studied best in the preparations stained by Van Gieson's method, since it selects the picric acid dye, and is readily traced as a slender thread in all sections where extreme atrophy has not been reached. The width of the rete layer varies considerably in different parts of the specimens. Over those portions of the corium where inflammation is actively present, with diffuse small round-cell infiltration and sclerosis, the Malpighian layer is flattened and drawn out, and the papillæ are broad and

FIG. 5.

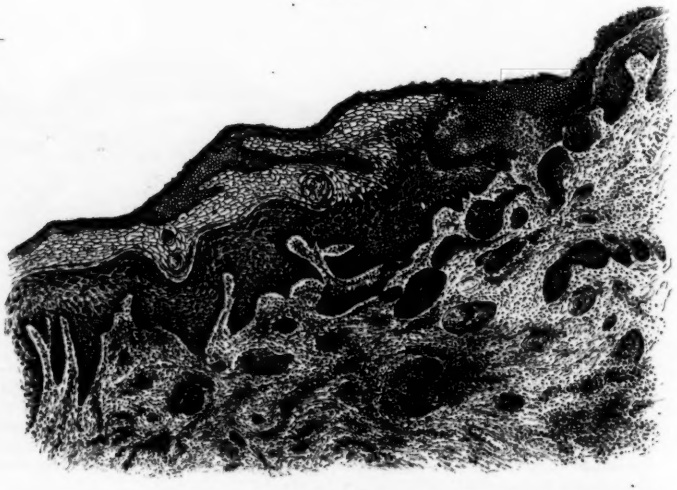


Showing hypertrophied epidermis, with blood-crust adherent to the surface; to the right fissure extending well into the corium. Hyaline transformation of outer portion of the corium, with entire absence of hair-follicles and sebaceous glands. In the deeper subepithelial tissue to the right, extensive areas of free red blood-corpuscles, with sclerosis of the elastic and muscular fibres; to the left, hair-follicles, bloodvessels, and bundles of muscular fibres in cross-section, with areas of small round-cell infiltration.

low, or wanting altogether. When, however, the inflammatory reaction is slight, small round cells appear, comparatively few in number, and the corium is but slightly altered; the papillæ are considerably elongated and more numerous, and long, finger-like processes of epithelium project between them. (See Fig. 5.) In all the fields where the extreme degree of atrophy has not been reached the individual cellular outlines in the Malpighian zone are sharp and distinct, pigment-granules are present in abundance, and the prickle cells are seen beautifully. The tissue beneath the epidermis furnishes us with a study of much interest. In some few places in the corium the delicate wavy outline of the elastic fibres is present, but in far the greater part a more or less extensive change is noted. The outer layers of the corium are

pale and slightly œdematous, the elastic fibres become indistinct, blend, lose their wavy outline, and become hyaline in character and matted together. Small round cells are found in large aggregations immediately beneath the epidermis, diffusely scattered throughout the corium and infiltrating the papillæ. In other places the corium appears dense and sclerotic, with scarcely any nuclei to be seen. The blood-capillaries have disappeared from the corium except in a few places, and no sebaceous or sweat glands can be found in any of the preparations. Areas of small round-cell infiltration extend deeply into the subcutaneous tissue, which also shows sclerotic changes. Here, also, hemorrhages have taken place, and free red blood-corpuscles are diffusely

FIG. 6.



Showing hypertrophied epidermis, adherent blood-crust on the surface; fissure extending through epidermis into the corium; and extensive round-cell infiltration in the subepithelial tissue. Hair-follicles and sebaceous glands entirely absent.

scattered through the loose stroma. Bloodvessels, fat, and unstriped muscle are present in normal amount. Points of special interest to be noted are the peculiar bundles that are found in the upper layers of the subcutaneous tissue. These can be seen both in longitudinal and in cross-section, and are well shown in the cut. (See Fig. 5.) While present in such numbers that it seems practically out of the question that they should be degenerate nerve-fibres, the histological resemblance is nevertheless most striking. The fibres are in distinct bundles, surrounded by dense fibro-elastic tissue, and on cross-section appear like nerve-fibres. The centre of each fibre is occupied by a dark round

body whose appearance is almost precisely like that of the axis-cylinder. That they are not nerve-fibres seems to be proven by the fact that the supposed axis-cylinder takes the hæmatoxylin color when stained by Van Gieson's method; that upon cross-section the fibres are not exactly round, but fit against one another like muscle cells; that upon longitudinal section spindle-shaped nuclei appear; and that when stained by Weigert's method the result is not characteristic. In none of the sections can any trace of nerve-fibres be positively found, though most careful search has been made for them.

The etiology of kraurosis is a matter upon which, as yet, the writers upon the subject have not been able to agree or to come to a positive conclusion. Breisky, who saw twelve cases, only attempted a clinical and pathological description, and could furnish no valuable etiological suggestion beyond the fact that some of the patients suffered previously from a vaginal discharge, and that in a number of them the symptoms of pruritus were markedly present.

Jenowsky[3] reports six cases, in one of which sclerotic œdema had been previously present. In his cases he considered that a long-continued discharge and syphilitic ulcers had had a definite bearing upon the etiology. This author compares kraurosis with leukoplakia of the mouth and tongue, and with urethral stricture.

Orthmann[11] made careful microscopical and bacteriological examinations of five cases from Martin's clinic, in Berlin, and while contributing to our knowledge of the pathology of the disease, could add nothing as to the etiology. He failed to find degenerate nerve-filaments in the diseased tissues, and considered gonorrhœa and a leucorrhœal discharge, one or the other of which had been present in all his cases, as etiological factors. He also examined the urine carefully for sugar, but could find no trace of it.

Hallowell[9] placed kraurosis among the large and interesting group of chronic inflammatory diseases of the skin and mucous membrane, such as ozæna, psoriasis of the mucous membrane, chimney-sweeper's eczema, and others, all of which are characterized by hypertrophy and metaplasia of epithelium, a hardened corium, and a tendency to terminate in epithelioma.

Olshausen[25] mentions the fact that in certain cases the atrophic changes have followed the removal of the uterine appendages. Others have also mentioned this fact, and have sought to associate it with the atrophy of the genital organs brought about by an artificially induced menopause.

It is quite possible that the climacteric period may play an auxiliary rôle in the progress of atrophy, but that this is an important factor is doubtful. Many of the cases have occurred in women past forty, and, on the other hand, a number of Breisky's cases were observed in women

who were pregnant, and Hallowell[9] describes one in a woman of thirty-three, whom he watched all through her pregnancy, and delivered at term without rupturing the perineum, although the skin surface of the vulva was extensively cracked and seamed.

Johnstone[20] describes kraurosis as trachoma of the urogenital canal.

Peters,[16] after careful microscopical examination, described the disease as a chronic hyperplasia of the subcutaneous tissue and corium, with a tendency to scar-like shrinking of the vulva. Attendant with and as a result of the subcutaneous atrophy, sclerosis and degeneration of the elastic tissue, the whole skin becomes involved with inflammatory œdema and atrophic lesions of the epidermis. But on the primary cause of the disease he throws no light.

Reed,[15] who re-introduced the subject of kraurosis five years ago, and presented a most complete description of six cases, together with the result of microscopical examination, suggested the term "progressive cutaneous atrophy of the vulva," on the supposition that the pathological lesions were confined to the skin.

As a result of the study of his own cases and the reports of others, he inclined to the theory that the disease of peripheral trophic nerve-filaments, or else the ganglia whence they originate, was the primary etiological factor. This, however, is a matter of theory and has no supporting evidence.

Longyear,[26] while advancing no positive opinion as to the etiology of kraurosis, nevertheless states that he does not believe the disease to be due to any local or constitutional disturbance outside the nervous system, and that the morbid process is due to defective nerve-action, probably reflex in origin.

Some writers have attempted to explain the origin of kraurosis in a previously existing specific lesion, but the large number of cases in which no syphilitic history can be traced, and in which venereal affections can be almost positively set aside, renders any serious consideration unnecessary. The testimony of Lewin,[22] too, who stated that he had treated between 70,000 and 80,000 women in his clinic at the Charité without ever seeing a case of kraurosis vulvæ, confirms the belief that this peculiar disease of the vulvæ is not of syphilitic origin.

Veit,[33] although himself having had but one case, reviews the whole subject in a scholarly article, and while exceedingly conservative in his statements as to what may be definitely proven in regard to the causation of the disease, nevertheless advances theories of etiology that are rational and deserving of careful consideration. In his opinion a close relationship exists between pruritus and kraurosis.

Recognizing in pruritus only a symptom, which by scratching is also able to produce an alteration of the skin of the vulva, in kraurosis he

holds that we have to do with an essential alteration of the skin, and accords to it a distinct position among diseases.

In his argument, Veit first calls attention to the fact that in the majority of cases of kraurosis the symptoms of itching are noticed at the onset, and then attempts to explain the precise relationship which exists between this disease and pruritus. While pruritus is but a symptom, definite lesions of the skin usually develop secondarily, owing to scratching and irritation, although actual lesions may be entirely wanting. Again, among the various causes of pruritus he recognizes that which later on develops into kraurosis. On the other hand, under the symptoms of kraurosis, he gives a prominent place to the itching of pruritus, which is only present at the beginning, but may be present all through the disease. So much only he feels is definitely proven. Hypothetically, however, he goes much further. Using the demonstration that the actual lesions of the vulva in pruritus are secondary, due probably to scratching or pinching up of the skin, he reasons by analogy that we can readily imagine that there was in the beginning a catarrhal inflammation of the vagina or the urethra, or a qualitative change in the urine; that these caused the sensation of itching, which again caused scratching, and again the peculiar alterations of the skin of the vulva, which anatomically are classed with the chronic inflammatory processes.

After the disease has continued for a long time the sensation of itching and the alteration of the skin may also continue even after the original cause of the disease has disappeared. The skin alterations eventually become to a certain extent permanent, and are curable only by excision. He feels, further, that he takes small risk in saying that with an intense degree of alteration of the skin, of small round-cell infiltration, *i. e.*, inflammation, there follows a marked shrinking, whereupon we have before us the picture of kraurosis. That all women do not complain of itching, or have not complained before, who seek relief for kraurosis, he easily explains by their sense of shame and the fear of being held in contempt by the physician as a masturbator, so that a failure to report this condition is quite excusable. Kraurosis, then, he describes as an ennarrowment and shrinking of the vulva, which is brought about by an inflammation of the skin, which again is brought about after intense itching, by scratching. That all cases of pruritus do not pass on into kraurosis is explained by the fact that many patients seek treatment for pruritus, and a sufficient alteration of the skin is not produced to bring about shrinking.

Sänger and Martin both lay stress on the interdependent relations which exist between pruritus and kraurosis, while Martin[7] places special weight on its development out of a previously existing inflammatory condition. Sänger alludes to it as a senile and presenile atrophy of the vulva.

After a careful review of the literature and a thorough microscopical study of our own case we would ascribe to kraurosis an inflammatory origin, modifying but slightly the theories of Veit and Martin. While it is possible that primary lesions in the trophic nerve-filaments, or the ganglia whence they spring, may be the cause of the local manifestation, this is purely a matter of theory, with no evidence whatever to support it, and seems to us unreasonable and not borne out by the pathological investigations. Our own opinion is that the cause is entirely a local one. Pruritus is such a constant symptom in the beginning of the disease in nearly all instances that its etiological bearing cannot be ignored, and we agree with the hypothesis of Veit, that the itching induces scratching, which in turn sets up a chronic inflammatory condition, with the formation of cicatricial tissue in the deeper layers of the derma and subcutaneous strata, shrinkage and contraction of the vulva, and atrophy of the skin surface. We, however, recognize the fact that the larger number of cases of pruritus apparently do not degenerate into kraurosis; that there is, therefore, some hidden impulse which causes further changes than ordinarily take place with the symptoms in this disease, with which impulse we are as yet unfamiliar, seems likely. This element may be either constitutional or local. When once this element is brought into play in the presence of pruritus we would go still further and be more explicit in our explanation as to how this result is brought about. As is well known, the skin surface is the constant habitat of various micro-organisms capable of producing inflammation and even suppuration. On rubbing, or scratching, irritation is produced and the normal cellular resistance impaired. The micro-organisms are now able to set up a low-grade inflammation which easily extends into the corium and underlying subcutaneous tissue. The skin surface may, however, be broken and the micro-organisms introduced from beneath the finger-nail or from the clothing. The corium and underlying subcutaneous stratum, rich in elastic tissue and unstriped muscular fibres, become sclerotic and hyaline in consistency, lose their elasticity, shrink and contract. The bloodvessels are pressed upon and their calibres gradually lessened and destroyed, while the nerve-filaments are rendered first hyperæsthetic and then succumb. Primary hyperplasia, the result of irritation, gradually gives way to atrophy, until the entire thickness of the skin above the areas of scarred tissue are involved. When the entire area has become atrophic the progress of the disease is arrested and the condition remains stationary, and the vaginal orifice narrow and contracted, but no further symptoms are manifest.

The atrophic process involves all the glands and appendages of the skin, so that the dry, glossy surface, dead-white appearance, and tendency to crack are readily accounted for. When this stage has been

reached it is readily seen that the only treatment that can be of any avail is the total removal of all scar tissue and an approximation of the edges of the healthy skin and mucous membrane.

The treatment of this condition has been divided into palliative and curative. The former is simply to relieve the subjective symptoms, which at times are excruciating. Carbolic acid and cocaine afford temporary relief, but the symptoms return soon after with renewed severity. Tait mentions the soothing effect of a solution of neutral acetate of lead in glycerin, placed on cotton between the nymphæ. Johnstone advises a salve of yellow oxide of mercury in the early stages.

Pure nitrate of silver applied repeatedly lessens the suffering, but has no effect upon the progress of the disease. Hot water often affords considerable temporary relief. Heitzmann has practised curettement and scraping of the diseased tissue, but the result has been entirely unsatisfactory and the process a long and painful one, requiring very many repetitions.

Martin[17] was the first to suggest complete excision of the diseased tissue as a curative measure, and applied the method in five cases, four of which were permanently cured, and one experienced a recurrence. Others have since followed the same method, the results obtained by Reed, and published in the article above referred to, being particularly gratifying. To be completely successful total excision of the diseased tissue should be accomplished. If this is done the prognosis is most favorable. Recurrence has occurred in some cases when this was not obtained and when the operation was performed before the process of atrophy had been entirely accomplished.

The history of the following typical case is herewith presented:

The patient, aged forty-three years, married nineteen years, is the mother of one child, which is fourteen years old. Her labor was entirely normal, no instrument being used and no febrile reaction following. She never had a miscarriage.

Until the last four years she has been a well woman, although always somewhat neurotic.

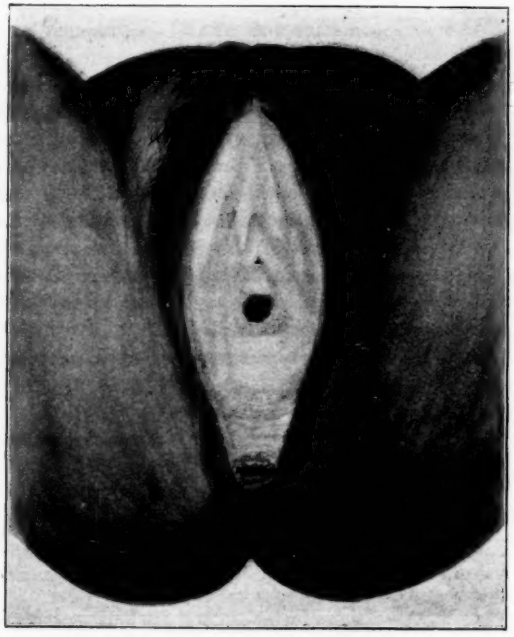
Four years ago she began to suffer from an obstinate itching about the vulva, which nothing could relieve permanently. Bathing with warm water gave as much temporary relief as anything, although all manner of washes and douches were tried by her physician. At times she suffered from a sensation of drawing and puckering about the vulva, which, however, lasted only a few hours, and then disappeared for a period of months. Her menstrual periods recur every four weeks, remaining for four or five days. The flow was free and accompanied by bearing-down pains which continued half a day. Some years ago she had a slight amount of leucorrhœa, but has had none whatever of late years.

At the present time she sleeps well and has a good appetite. She suffers somewhat from indigestion, resulting in gastric distress and abdominal distention after eating. Her bowels are regular in their action.

Urination at times is frequent, otherwise normal. There is a strong family history of pulmonary tuberculosis. Any possible venereal taint is easily eliminated. She is extremely nervous and excitable.

The patient was referred for treatment for an obstruction at the mouth of the vagina, which had been gradually increasing for some three or four years, and which had rendered sexual intercourse more and more difficult, until about two years ago, when it was abolished, not on account of any particular pain it caused, but because penetration became a physical impossibility. The contraction continued to such an extent that the husband and wife, as well as the physician in attendance, feared it might eventually become so complete as to prevent the escape of the menstrual flow. It was thought that possibly a tumor developing in the vagina might be the cause of the obstruction.

FIG. 7.



Showing affected area, with contracted vaginal orifice.

Repeated attempts at dilatation had been made by the physician in attendance, but all had resulted unsatisfactorily. The contraction progressed steadily. (See Fig. 7.) An examination of the parts affected disclosed the following condition:

Beginning in the mons veneris half an inch above the clitoris, and extending downward (in the form of an elongated oval) to just below

the anus, the skin and muco-skin of the entire enclosure and perineum presented a dead-white appearance, almost as though it had been badly macerated.

All trace of the clitoris and the nymphæ had disappeared, and the labia majora were almost obliterated as such. No trace of the fourchette was found, and the vaginal opening was barely sufficient to introduce the tip of the little finger, and only then with considerable pain to the patient. At the vaginal opening the disease extended laterally from the vaginal mucous membrane for the distance of one-half to three-quarters of an inch. There were no local points of redness and no leucorrhœal discharge.

At this time the patient had no symptom whatever except the obstruction, and that, of course, was noticeable only when coitus was attempted. The itching and drawing feeling, which at no time had been prominent, had disappeared. There was no obstruction to the flow of urine nor to the passage of the bowels, although the disease extended completely about both the urinary meatus and the anus. Surgical treatment was decided upon, and the patient submitted to the following operation, with the object of obtaining a sufficiently patulous condition of the vulva to enable her to resume her marriage relations:

A strip of skin one-half to three-quarters of an inch wide, beginning at the level of the urethra on one side and extending around the complete circumference of the vulva to the level of the urethra on the opposite side, was excised. The innermost edge of this strip followed the line of juncture of the vaginal mucous membrane throughout, there being a fairly sharp line between the healthy vaginal mucous membrane and the diseased tissue outside. Laterally, the strip was sufficiently wide to include all the diseased area, but as the incision circled around onto the perineum, between the vulva and anus, the strip, although it widened, could not be made to include all the diseased area, as this extended to below the anus. The incision was, however, carried as near the margin of the anus as possible.

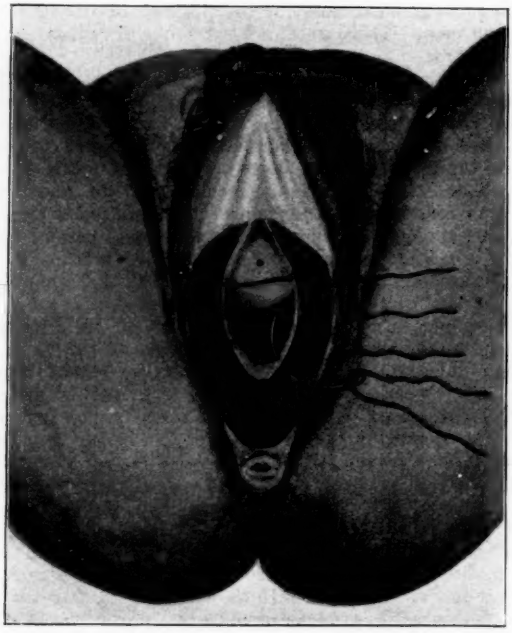
The knife was now passed directly into the septum between the vagina and rectum, through the denuded area to the depth of an inch, and the tissues freely incised laterally, with the object of cutting the muscles. The effect of this was to produce practically a partially torn or relaxed perineum, with a more or less gaping vulval orifice. The cut edge of the mucous membrane of the vagina and the skin were now readily brought together by interrupted silkworm-gut sutures, supplemented (see Fig. 8) by a continuous suture of fine silk. The object of this double suture of fine silk was to secure an accurate approximation of all incised edges, and to ensure the more certainty against any tearing out of the sutures when the vaginal plug, which it was proposed using, was inserted in the vagina.

A large glass plug two inches in diameter was then passed into the vagina and held in place by a T-bandage. The following day, on account of the tension of the stitches, this plug was replaced by a smaller one.

The plug was removed twice daily, and vaginal douches of boric acid administered each time. Union of the stitches being completed, they were removed at the end of ten days. The bladder was catheterized each time the plug was removed. Gradually the patient was taught how to introduce the plug, and on returning home at the end of

three or four weeks was given a hard-rubber plug one inch and a quarter in diameter, with instructions to introduce this twice daily, lie down, and allow it to remain an hour each time. After several months the introduction was reduced to once a day, and was finally withdrawn altogether. Just before allowing the patient to omit the daily introduc-

FIG. 8.



Showing denuded area and approximation of healthy skin and mucous membrane.

tion of the vaginal plug a letter was received from the husband relative to some irritation about the urethra, which was evidently set up by the frequent introduction. We quote the first few lines of this letter:

DEAR SIR: I wanted to write to you before now, but put it off from time to time. As far as sexual relation is concerned, Mrs. ———'s operation is a perfect success . . .

Yours truly,

The whole subject of future contraction is open to discussion, but we feel confident that such will not take place in this instance, for the disease had apparently reached its height, and had come to a standstill. But whether or not this be true, all the diseased area laterally was dissected away, and healthy skin and healthy vaginal mucous membrane

united. It is true the tissue about and above the clitoris is involved, as is also that about and below the anus, but it will be impossible for any amount of contraction at these points to affect the vaginal opening. The only danger to be feared will be the future involvement of the now healthy vaginal mucous membrane and skin, which have become united.

The patient will be watched with great interest. The possible future contraction of both the urethral and anal openings has been considered. Should either or both occur, a similar line of treatment to that carried out in the case of the vaginal opening will be adopted.

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THE IMMEDIATE AND REMOTE EFFECTS OF ATHLETICS UPON THE HEART AND CIRCULATION.[1]

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During the past six years I have had frequent opportunities of examining athletes competing for positions and playing upon the various teams of the University of Pennsylvania, and have made studies, as far as was possible under the circumstances, to determine the effect of athletics upon the size and efficiency of the heart and the character of the circulation. It has not been possible for me to carry out elaborate investigations into the manner of production of the various phenomena which attend exercise, and I shall not enter upon any discussion of this aspect of the question now. In outlining the heart of patients or athletes, I follow a method more or less like that used by Sansom. After carefully mapping out the heart's borders by percussion and auscultatory percussion, I mark these borders with a brush dipped in oil, and similarly mark the middle line of the sternum, indicating the suprasternal notch by a V-shaped figure at the top, the ribs (by broken lines), and nipple and position of the cardiac apex by a circular and a crossed figure respectively. A piece of soft tissue-paper is then pressed over the chest, and in this way the outlines are secured in their exact size. The tracings which I present here are exact copies of such outlines upon heavier paper.[2]

In watching the effect of training upon the heart, my attention was first directed to the ease of development of a systolic murmur over the heart. This occurred in the late summer of 1893, when I spent some time with the candidates for the football team during their preliminary training. The men who were candidates had for the most part been resting, and were, therefore, out of condition. I noticed that a systolic murmur over the body of the heart was not unusual, and in a special trial of nine of the candidates I found that three promptly developed a very evident murmur of the kind after a little exertion. During the subsequent fall term I tested the same men while in actual practice, and found that the murmur, before so readily produced, did not show itself.

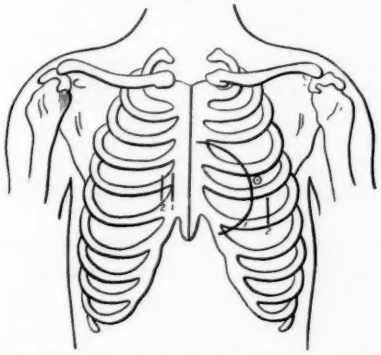
This led me to study the conditions of the heart immediately before and after exercise more carefully, the method employed being to map out the borders of the organ as already detailed just prior to some athletic sport, and again immediately after the exercise.

[1] Read at the meeting of the Association of American Physicians, May, 1899.
[2] The cuts are reduced representations drawn to scale.

Of a number of charts a few may be taken as typical. The two I exhibit first were prepared from young men rather new at athletics.

Chart I. was taken from a boy aged nineteen years who had not previously trained for any form of sport, and who was now engaged in one-half mile running. The chart exhibited represents the position of the

CHART I.



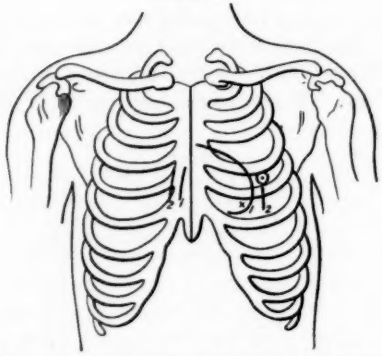
Illustrating position of the right and left borders of the heart before (1) and after (2) running one-half mile.

left and right border of the heart before and after a trial run of one-half mile. Before the race the sounds at the apex and over the body of the heart within the apex were normal, excepting for a certain deliberateness of the first sound. The pulmonary sound was, perhaps, a little accentuated. There was a suspicion of murmurishness of the first sound toward the base of the heart. After the race the apex-beat was quick and circumscribed, and there was a distinct, diffuse thrill over the apex and in the epigastrium. Auscultation discovered a slight systolic puff, and the second pulmonary sound was distinctly accentuated. The murmur was localized in the region of the apex.

Chart II. was taken from the chest of a young man aged twenty-two years, a half-mile runner. Before the race, at which time this chart was prepared, the heart-sounds were found to be normal, though perhaps a little quick and irritable. After the exercise a distinct systolic thrill was found at the apex. The first sound of the heart was murmurish, but the relative values of the first and second heart-sounds at the pulmonary area were retained. These two charts illustrate very strikingly the effect of exercise upon the heart in comparatively untrained young men.

As contrasts to these I select the next two, which were prepared from trained athletes.

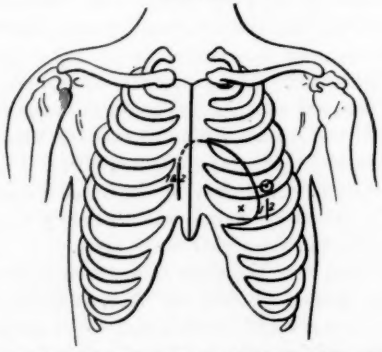
CHART II.



Borders of heart before (1) and after (2) one-half mile run.

Chart III. was taken from a walker, aged about twenty-three years, who had engaged in active athletics for a number of years, and who was unusually sturdy, walking in remarkable time a mile or more, without any of the evidences of fatigue so commonly observed after this sport. On the occasion on which this chart was prepared, Mr. F. showed at the preliminary examination a strong and circumscribed

CHART III.



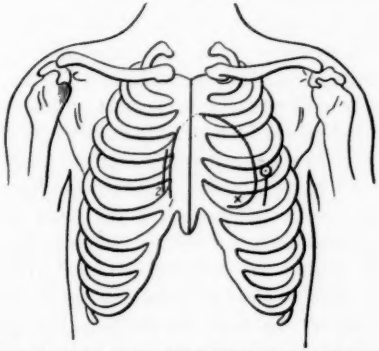
Borders of heart before (1) and after (2) three-quarter mile run.

apex-beat, with a long and very muscular first sound, but without a murmur. At the pulmonary area both sounds were distinct. There was a suggestion of a thrill on palpation over the apex. After walking three-quarters of a mile in very rapid time the apex-beat was found to be violent, and on palpation there was a rather more distinct thrill. The first sound was sharp and strong, but muscular, and there

was a puffing systolic murmur heard over the pulmonary area, and accentuated by breathing, but the second sound was not particularly accentuated in the pulmonary area. The chart is mainly interesting in showing the right border of the heart exactly in the same position before and after exercise, though the entire organ seemed to be somewhat hypertrophied.

Chart IV. is that of a young man of remarkable physique, the holder of three world's records. The chart exhibited was taken after sprinting and hurdling. Prior to the exercise there was a systolic murmurishness at the apex, but no distinct murmur. The pulmonary second sound

CHART IV.



Borders of heart before (1) and after (2) sprinting and hurdling.

was about normal. The aortic second sound was a little accentuated. After the exercise there was a suggestion of a thrill in the region of the apex, and possibly also in the epigastrium. There was no murmur, but the pulmonary second sound was a little accentuated. In this case the right border of the heart was little affected by the exercise.

These charts are sufficient to illustrate the changes in the outline of heart as obtained by percussion and auscultatory percussion after exercise. I cannot now enter into a discussion of the causation of these changes, but should like to refer briefly to one or two points of interest. It has been demonstrated by physiological experiment that muscular exercise causes a fall in the blood-pressure in the peripheral vessels, and probably a dilatation of these vessels. Under these circumstances we should not expect an increase in the size of the heart, and increased intracardiac pressure as a result of strain. It does seem, however, that in the beginning of exercise there is a temporary increase in pressure at the centre. It has long seemed to me that the phenomenon known among athletes as "second wind" was to be explained by the recovery of the right ventricle after the preliminary overdistention incident to

exertion. I had not seen this view expressed until within a few days, when my attention was called to the recent article by Dr. Allbutt, in his *System of Medicine*, vol. v. I quote from this article (p. 849): "The dilatation is, I think, concerned in 'second wind'; the healthy heart increases its out-put, the lungs expand, resistance falls, the right ventricle pulls itself together, and second wind is established. This process, trying enough to an unsound or defective heart, to young boys and to elderly men, is to the healthy heart of comparatively young adults perhaps never injurious." This expresses my view of the case exactly, a fact which is all the more gratifying to me because Dr. Allbutt's views are based upon observations of under-graduates at Cambridge, as mine have been upon students in Philadelphia. It seems to me not unlikely that the size of the right ventricle depends upon the degree of recovery of the right heart after the first distention. I cannot, however, agree entirely with Allbutt in the statement which follows immediately after what I have quoted: "I have many times seen under-graduates and others look ghastly at the end of a long spurt of exercise, but I never saw a sound young man the worse for a temporary stress of this kind; if, as in a few cases which I have seen again and again in growing youths, dilatation of the heart occurs, leading to cyanosis, panting, confusion, or vertigo, this oppression is generally sufficient of itself to stop the exercise in time." Of course, all of us who have had occasion to observe athletic sports of any kind have seen instances of the sort alluded to in the sentence just quoted, but I have seen a few cases in which recovery was not as prompt as is intimated, and in which, though the normal condition was restored after a short interval, it seemed to me that some sort of permanent damage must have been done, though I could not demonstrate it. I may cite, for example, the case of a young man playing football, who was in superb physical condition, and had never been hurt or disabled in any way, and who, on the occasion of a trying game, both physically and mentally, suddenly collapsed toward the end of the game. I had repeatedly examined him prior to this time, and within a day or two had outlined his heart and other organs. I saw him immediately after his attack, and found him bleeding at the nose, more or less cyanosed, and with a weak, fluttering pulse. On examination of his chest I discovered that both sides of the heart had become enlarged, the increase in size toward the left and right being fully three-quarters of an inch or an inch. The man was prostrated, and did not recover any sort of control of himself for some hours. He then became rapidly better, and after a few days was seemingly quite well. He did not engage in football for ten days, but at the end of that time resumed practice and regular play without any apparent evil consequences.

A case of similar nature is the following:

W. R., aged nineteen years, weighed 122 pounds during the winter of 1897. During the spring and summer he rode a bicycle excessively, frequently riding twenty-five miles in the evening after his day's work. His weight declined to 109 pounds. In August he went to the seashore, and while bathing his strength was overcome by too much exertion at swimming, and he began to expectorate blood. The next day, and during several days of the following week, there were repeated hemorrhages of the same sort. The total loss of blood was not very great, however. There was at the same time shortness of breath and cough on the very slightest exertion. The heart became intermittent and very tumbling in action, and both sides increased in size; the left border being in the nipple line, the right border beyond the right border of the sternum. There was no murmur even after exertion, when I examined him some time after the onset; the second pulmonary sound was a little accenuated. In this case, of course, the patient had not been in perfect health prior to his overexertion, for he had been progressively overtaxing himself during the spring and summer.

Another instance of a similar character occurred in a patient, J. A. F., a young man, aged twenty-four years. A year before he came under my observation he had taken a long bicycle ride, when he was not in a good physical condition, and the symptoms began immediately. He had been working in an office and was not in a good condition when he undertook a four days' ride on a tandem bicycle, the minimum daily distance being fifty miles and the maximum being seventy miles. He was prostrated at the end of the four days. His heart became very irregular, and he was confined to bed for four months. At the present time his heart extends beyond the left mammary line, and on the right to the border of the sternum. There is a suggestion of a systolic thrill and murmur at the apex, but no positive signs. The second sound at the base of the heart is not accentuated.

These instances are sufficient to show that rather unfortunate results may occur from violent excessive exercise in young persons not in proper training; and even at times in those well trained. At the same time, the charts I have presented illustrate the fact that there is a decided difference in those who are carefully and those who are insufficiently trained. With Dr. Da Costa, who discussed this question many years ago in his Toner lectures, I should be most unwilling to see danger in athletics unless the proof were positive. There is, of course, a certain amount of danger in every sport, but the danger in athletics is certainly not very great, and the necessity of careful training, not only for the combats of the athletic field but for the general struggles of life, is demonstrated by my observations.

Alluding now to the question of the auscultatory sounds over the heart, I recall the fact that in numerous instances I have discovered a systolic murmur over the body of the heart at the pulmonary area and at the apex. It is by far most common to find this at the pulmonary area, and in young boys it seems almost habitual. This murmur is usually attended by a pulsation over the pulmonary area, and on care-

ful percussion I have found that the oblique border of the heart was somewhat convexly curved toward the left. From these facts and other considerations I have become convinced that the cause of the murmur is to be found in a dilatation of the conus arteriosus of the pulmonary artery. The murmurs at the apex may undoubtedly be due to mitral regurgitation, but I cannot convince myself that all found in this situation are of this nature. Some, I believe, are intra-ventricular; others, possibly cardio-pulmonary in origin.

With regard to the second point in the heading of my paper—viz., remote effects of athletics—I can speak only of the remote effects in the sense of the results following at intervals of a few months or years. I believe that there is also a possibility of development of symptoms many years after cardiac strain without any indications in the intervening time. We all recall the interesting publication of Dr. Morgan on the *University Oars*, being a study of the after-health of men who rowed in the University boats of Oxford and Cambridge between the years 1829 and 1869. I would also direct attention to the Toner lectures of Dr. Da Costa, delivered in 1874, in which he called attention to the possibility of strain as a result of baseball. In my own experience I have found a number of instances of distinct symptoms of cardiac disturbance in football players who had devoted themselves rather steadily to sports during their collegiate life. As cases in point, I would briefly refer to the following notes:

C. S., aged twenty-five years, played football steadily at his preparatory school, at college, and during his university years, and baseball less regularly during the same time. After his graduation and his entrance upon active duties, he complained of a feeling of oppression in his chest, which he described as a sensation of distention, as if the heart was too large for the body. Sometimes there was palpitation and nearly always a consciousness of the heart in some form or other. His heart extends as far as the mammary line, and to the right border of the sternum, but there is no murmur or any sign of valvular trouble. He finds that the use of tobacco and alcohol, in which he has always indulged moderately, are prone to produce unpleasant over-action of his heart.

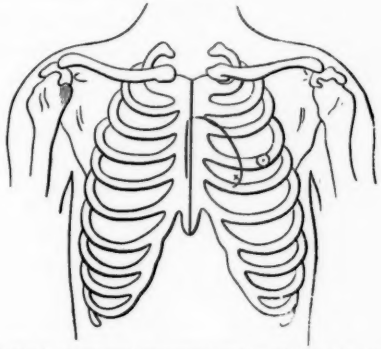
H. W., a football player and oarsman, steadily followed athletics during his college years. He now complains, after a few years of cessation from athletics, of symptoms similar to those just described. In particular he has noted, from time to time, a tumbling sensation in the heart-beat, and he sometimes feels a sensation of oppression or dyspnœa.

C. G., a football and baseball player during six years, has complained, since his cessation from athletics, of vague oppression, fulness in the chest, and some dyspnœa. He has, however, grown distinctly stouter in the two years during which he has relinquished exercise.

These cases will suffice to illustrate the kind of symptoms met with. I have seen many instances of this sort, and in seeking to explain their

causation, have naturally been interested in the condition of the heart itself. Charts V., VI., VII., and VIII. are selected to show the increase in the size of the heart, resulting from several years' athletics. Others

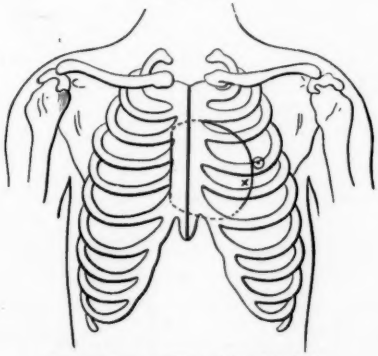
CHART V.



Football player and oarsman; outline of heart in 1894.

might be presented to illustrate the same point. It will be observed that in the two cases from which the charts were prepared the right and left ventricle increased decidedly in size during the three or four years that elapsed between the first and the second examination. In each of the men from whom these charts were taken symptoms of the sort referred to were found.

CHART VI.

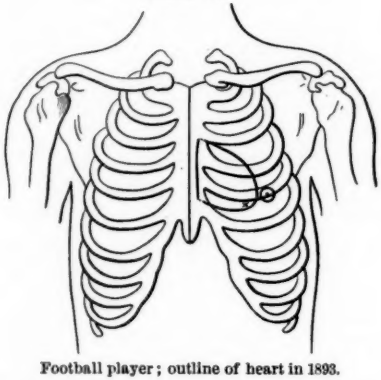


Same person; outline of heart in 1898.

Allbutt, in the article before alluded to, in mentioning the after-effects of athletics, suggests that the mean blood-pressure increases as a consequence of the withdrawal of the reducing influence of exercise.

I have not been able to assure myself that the blood pressure was actually lower, as I have found the sphygmomanometer an uncertain instrument, and I cannot bring myself to trust in the evidence afforded by direct palpation of the artery. I am, however, quite ready to believe

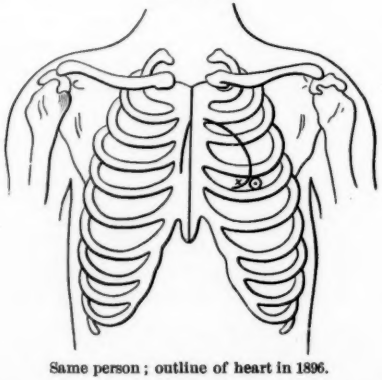
CHART VII.



Football player; outline of heart in 1893.

that the blood-pressure is increased on account of the evident hypertrophy, if for no other reason. In athletes who have ceased to indulge in sports, and who have for some years suffered with symptoms of oppression, there seems to be a decline of the symptoms mentioned after

CHART VIII.



Same person; outline of heart in 1896.

some years and a return to more normal conditions. I am led to make this statement from the history obtained from individuals in middle life who had been athletes during their college years. My experience has not, however, been long enough to follow such cases from the beginning,

and I hesitate to make assumptions in cases which I had not the opportunity of examining in the beginning. On the other hand, observations which I have had the opportunity to make in men past middle life who had been very athletic in youth, have led me to believe that muscular disorders of the heart may arise after middle life as remote consequences of overexertion in early manhood. I do not speak at length of these because they are cases in which I have no means of knowing the conditions in the intervals between the beginning and the final trouble.

From the observations that I have been able to make it seems positive to me that overdistention of the heart is frequent in athletic contests, and may cause harm if the individual is in ill-health or badly trained; and, secondly, that hypertrophy and symptoms of overaction sometimes result from continued athletics. As practical conclusions, I would suggest rigid medical supervision of college athletics; and, secondly, that young men be encouraged to continue some form of exercise after discontinuance of regular athletics.

A SHORT UMBILICAL CORD AS A CAUSE OF DYSTOCIA.

WITH A DESCRIPTION OF A NEW SYMPTOM.

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An unusually short umbilical cord has long been known to possess inheritant difficulties. Early in this century the classical Denman[1] and the observant Jorg[2] not only described the dystocia due to an abnormally short cord, but gave directions for the conduct of a labor under conditions in which this rarity presented itself, and their advice has not been improved upon since their day. Obstetricians draw a distinction between the cords which are inherently short and those which are shortened by being wound about the fœtus. The former is known as a "natural" or "absolute" shortening, the latter as a "relative" or "accidental" shortening of the cord. This paper deals with the former or absolute variety.

The relative length of shortness of the umbilical cord cannot be fixed. A cord which would be of normal length, or would at least allow of easy, unassisted birth in one case, would, in another, be too short to permit a fœtus to be born without help. The cord may vary in length from two inches to many feet. To be a factor in the delay of labor the cord must actually be so short, measured from the umbilicus to its placental insertion, that the fœtus cannot be born without one of several accidents

being likely to happen. These may be: rupture of the cord at any point along its course, detachment of the placenta, inversion of the uterus, or, as has not previously been clearly pointed out, an umbilical hernia in the child. The first two accidents are the more common ones, the last two the rarest, in the presence of this condition.

The location of the placenta is vital in determining the absolute length or brevity of the cord. If the placenta lies low in the uterus a cord which would prove too short were it higher in the uterine cavity is sufficiently long to permit the birth of the fœtus. A central or marginal insertion of the cord into the placenta must also be taken into account in estimating its actual length. This is, of course, not possible before birth.

In the consideration of the accidents which may ensue from an unusually short cord it is necessary to give some thought to the tensile strength of the cord and the influence of the expulsive forces upon its elasticity. Connective tissue acquires its tensile capacity from the amount of elastic fibres it contains. In the mucoid or colloid structure of the umbilical cord, elastic fibres are found increasing in number as the end of pregnancy advances. The younger the cord, the fewer and weaker are the elastic fibres—Hertwig.[12] Lesshaft,[3] quoting the figures of Valentin and Rauber, states that the tensile strength of connective tissue containing elastic fibrils is greater than that of a wire made of lead and equal to that of a cord of tow. The elasticity of the umbilical cord has also been made the subject of study by Duncan[4] and Lamare.[5] The former observers found that the average cord breaks under a strain of eight and one-quarter pounds, while Lamare's figures are somewhat lower than this. He found that the cord ruptures under the influence of a falling weight at about five and one half pounds. It seems to me that the statements of Duncan and Turnbull are entitled to greater consideration, since their experiments were conducted on principles more nearly consistent with the actual conditions.

Assuming, then, that the average cord will break under a strain of between eight and nine pounds, and that the force of the uterine contractions during expulsive pains is forty pounds, as given by Herman,[6] it is evident that the cord must be long enough during birth to allow the umbilicus to reach the vulva and slightly beyond it, or it will break, or will tear the cord from its umbilical insertion, or will tear the placenta from its insertion, producing hemorrhage, death of the fœtus, or an inversion. Negrier[7] gives the average length of the parturient canal at the time of the expulsion of the fœtus as 8¾ inches, and I am not acquainted with any figures which are more reliable. The limit of safety, then, in the length of the cord would be about ten inches—that is, a cord shorter than this would be almost certain to be productive of accident. Kaltenbach[8] points out that the placental insertion of the

cord has much to do with the dangers of its shortness, as we have already seen. He believes, however, that if the placental insertion of the cord is at the fundus, 35 centimetres (13 inches) is too short for a safe delivery, while in the lower insertion, he thinks 20 centimetres (8 inches) long enough for easy birth. So far as I can learn, Kaltenbach stands alone in his belief that even under favorable conditions a cord of 8 inches is a safe one. On physical grounds we have seen that the cord should be at least 10 inches long to ensure an unassisted delivery.

Cords shorter than 10 inches are very rare. In two services which embraced upward of 1200 cases I never saw one. Dewees[9] had never seen an absolutely short cord, and was inclined to doubt the existence of such an anomaly. Many ruptures of the cord, some cases of inversion of the uterus, some accidental hemorrhages from the placenta, so-called, and some of the umbilical hernias in children, as I shall show, can be traced to this phenomenon.

Before referring to the diagnosis and treatment of an absolutely short cord I shall record a case which came under my observation, in many respects a classical one:

Elderly primipara; position, L. O. A.; protracted first and second stages; short cord; separation of the placenta during labor; post-partum melancholia; umbilical hernia in child. Mrs. E. G. B., aged thirty-four years, married eight years, primigravida. Menstruation has always been regular but painful. There is no family or personal history of importance. There is no history of syphilis. The last menses appeared February 22, 1895. The predicted date of confinement was November 29, 1895. The pregnancy was uneventful. The urine was negative throughout. The feet and hands were somewhat swollen toward the end of the gestation, but there was no general œdema. The patient had read some popular work on marriage, and was very apprehensive as to the outcome of her labor. During her confinement she talked in an absurdly mistaken manner of child-bed fever, second-stage pains, child's stomach-rash, etc. The patient was a stout woman, but not inordinately so; she was phlegmatic, stupid, and exceedingly irritable. The pelvic measurements showed a pelvis somewhat larger than usual.

On December 3, 1895, she had a few false pains, with the discharge of some fluid, which I had no opportunity of seeing. At 8 P.M., December 6th, labor began with infrequent and weak pains, and the patient remained in this condition until 1 P.M. of December 7th, when she complained greatly of fatigue, and begged to have the child taken from her. She was given a hypodermatic of one-quarter of a grain of morphine, after which she slept for two and one-half hours. At 5.40 P.M., labor pains again started in, weak and infrequent. The fœtal heart was then beating at the rate of 140. At 10 P.M. the patient was given fifteen grains of the muriate of quinine. In half an hour the pains had become much stronger and more frequent. The cervix had almost disappeared, and at 11.30 P.M. the membranes were artificially ruptured, in the hope of hastening delivery. It was, however, 2 A.M., December 8th, before the patient was fully in the second stage. The pains were now frequent and expulsive, and again and again the head could be seen at the vulva, only

to be retracted as soon as the uterine contraction ceased. In the intervals between pains, as well as during a pain, bright arterial blood constantly escaped, but not in sufficient quantity to cause alarm or to weaken the patient. During the entire second stage the patient complained of pain over the left side of the uterus, at a point where I subsequently located the placenta. She urinated frequently during the beginning of the second stage, *between pains*, passing a small quantity of urine at a time. At 5.30 A.M. a male child, weighing six and three-quarter pounds, was born, the head being peeled out of the labia, and firmly held, despite the efforts of retraction, which ceased when the biparietals were in the vulva. The child was somewhat asphyxiated, having passed meconium for twenty minutes previous to its birth and being covered with it. It was easily resuscitated, the cord being tied at once. At no time did the necessity of applying forceps seem necessary to me.

The hand placed over the fundus immediately after the birth of the child could easily feel the placenta at its insertion on the left side of the uterus about midway between the fundus and the cervix. It was expelled with such ease twenty minutes later that I was convinced at the time that it had been lying loose in the uterus, an opinion which was confirmed by finding its maternal surface covered with a large and moderately thick clot. This condition also made clear the source of the hemorrhage of arterial character during labor. The cervix, I might add, had not been torn. The placenta was the seat of marked calcareous degeneration The cord measured, in its entirety, ten and one-half inches.

Convalescence was interrupted by a mild melancholia, which disappeared in two weeks. The child was noticed the following morning to have a marked protrusion at the umbilicus, which, after the dropping off of the cord, presented a granulating surface. A month later it looked like a small collar-button. Under the application of the solid silver stick and a compressing bandage it disappeared in four months. This protrusion was at first thought to be a Meckel's diverticulum, but a careful subsequent examination proved it to be an umbilical hernia. I believe that it was the direct result of the tremendous tension exerted upon the cord and its attachments at both ends during labor.

The delay in both the first and second stages can be credited without harshness, I believe, to the extreme brevity of the cord. The fact that the patient was an elderly primipara, who was somewhat stout, may have been an element of delay in the first stage, but it would be going far afield to attribute all the symptoms—the pain over the placental site, the arterial bleeding during labor, the recession of the head between pains, the frequent urination between pains, the umbilical hernia of the child—to anything but the fact that the cord was abnormally short, so short, in fact, that the child could just be born without artificial assistance.

SYMPTOMS AND DIAGNOSIS. The symptomatology which characterizes the absolute or natural short cord does not differ much from that which is seen when the cord is shortened by being coiled about the child's body. The variations which may present themselves in a perfectly normal confinement may easily obscure the symptoms of a condition at

no time common. Probably the most characteristic phenomenon to be attributed to the short cord is[1] the recession of the head with the dying out and disappearance with each successive pain. This is so marked, and so much greater than the recession which follows the usual cessation of uterine contraction during labor, that it attracts the attention of the accoucheur at once. The entire parturient canal diminishes in length with the advent of uterine pains, increasing again as the uterus relaxes, so that in an uncomplicated case the head naturally recedes after a certain advance. When, however, in addition to the natural recession there is added the elastic pull of an umbilical cord which will not permit the fœtus to maintain the advance it has made, it is evident that not only will the head recede with the end of each pain, but that this recession will be so rapid and so constant that it will offer a striking feature in the delay of labor. In the case narrated this recession was a puzzling factor to me, for I confess that its cause was not clear to me before the child was born.

Those who have written on the subject give a prominent place in the symptomatology to[2] pain over the placental site and depression of the placental area during a pain or during traction by the forceps, attributing the pain to peritoneal stretching. (Duncan.[4]) The former of these elements was decidedly in the foreground in my case. The latter was not observed. It is natural to expect pain in this condition when the placenta is undergoing a pull or a tug with each succeeding contraction of the uterus, for, though the entire genital canal diminishes in length with each pain, the child advancing despite the shortness of the cord, the tendency is for the placenta to become freed from its attachment to the uterine wall. Actual detachment occurs when the cord is exceedingly short in all those instances in which the umbilical insertion does not give way. In my case the placenta undoubtedly suffered partial separation, a deduction easily made from the constant appearance of bright blood during the labor, and from the condition of the placenta after its delivery. The fact that the recession of the head ceased upon the birth of the biparietals in my case was also unquestionably to be attributed to the loosening of the placenta and the consequent absence of further resistance.

Bleeding during labor, which does not come from a placenta prævia, or a torn cervix, or a tear in the vagina, should always direct suspicion toward the placenta, and when there is a combination of symptoms such as are included in this description the cause may not unfruitfully be sought in a shortened cord. In the case presented the hemorrhage was of an arterial character throughout, and aroused curiosity as to its probable source. It was present as well in the absence of pains as when the uterus was contracting. At no time did it impair the volume of the pulse of the patient, who was rather a plethoric woman.

It might well be expected in a condition in which there is a constant opposing influence on the accession of each pain to the advance of the fœtus that an exhaustion of the pains would ensue as a consequence. In my case this did not occur during the second stage, in which the dystocia was most marked. The delay in the first stage I have been inclined to attribute in part to the fact that I had an elderly and rather stout primipara to deal with. Napier,[10] however, regards uterine inertia as a cardinal symptom of a shortened cord or of one absolutely short. The expulsive pains of the second stage in the case here reported gave no indication of abnormality alone—they were strong and frequent Another symptom which was lacking in my patient was one upon which much emphasis is laid by King,[11] who has reported a number of these cases. He assumes that it is indicative of the patient's natural desire to obtain the benefits of gravity in delivering herself of her child, a view in which he was anticipated by Denman.[1] It certainly seems most natural for a patient with this anomaly to assume the upright position, and, as a matter of fact, my patient was frequently in the semi erect position during the second stage, but made no mention of the fact that that posture gave her any relief.

A symptom which I wish to add to the symptom-complex of a natural short cord—which might be equally applicable to a cord wound about the child's body, and which I have been unable to find elsewhere recorded—is one that was quite striking in my case—the frequent urination during the first part of the second stage. A moment's consideration of the anatomy of the soft parts at this stage of labor will aid, I think, in giving a clear notion of the significance of this symptom.

The bladder lies entirely above the pubes when the second stage has become established. As this stage proceeds to its natural termination by the advance of the fœtal head the bladder becomes tightly pressed against the symphysis, lying parallel with it. The urethra, too, is compressed and becomes elongated. It is notorious how difficult catheterization becomes at this stage of labor, and how almost impossible it is for a woman to urinate spontaneously at this time. Urination, then, on the disappearance of each pain, when the cervix had entirely disappeared, was a striking phenomenon, and is to be accounted for only on the ground that as the expulsive effect of a uterine contraction wore off the short cord was sufficiently strong to draw back the body, and with it, of course, the head. This manœuvre allowed the bladder again to fall forward and to empty itself of its contents, being stimulated to the act by its frequent compression. As soon as the head had advanced to a point from which the retractive efforts of the short cord could no longer dislodge it the urination ceased.

It is a perfectly logical deduction, I think, that when we have the unusual symptom of urination during the intervals of pain in the second

stage, especially at its beginning, that the attention of the accoucheur should be directed toward the shortness of the cord as the probable cause of this unusual clinical feature. He should be especially suspicious of this condition if there are a simultaneous recession of the head in the intervals, arterial bleeding almost constantly, and one or more of the other symptoms already recorded.

Briefly to summarize the diagnostic points in the order or their importance, I would place them as follows:

1. Recession of the head in the intervals of pains.
2. Arterial bleeding during and between uterine contractions.
3. Urination in small quantities in the intervals of pain after the establishment of the second stage.
4. Pain over the placental site, especially during a uterine contraction or during the application of forceps.
5. A desire of the patient to sit up.
6. Uterine inertia.

TREATMENT.—It is unfortunate that a diagnosis of an absolutely short cord can rarely be made with certainty before the birth of the child as far as the umbilicus. If it were possible always to make sure of the condition treatment would be of little avail. When a short cord is suspected, and the life of the child is thought to be in danger from the objective signs of an accelerated heart or the passage of meconium, the labor should be terminated as soon as possible. By pressing the fœtus down the placental insertion of the cord is brought near its umbilical insertion, and the danger of rupture somewhat obviated. But this is not always feasible or possible. Postural methods may be tried by having the mother assume a squatting or kneeling position, as recommended by Denman[1] and praised by King.[11] If the child presents by the breech, the cord should be tied as soon as the umbilicus is born. The advice given in some text-books, regardless of the position, to tie the cord if it is found too short to allow the birth to take place, is little short of absurd. The impossibility of tying the cord of a child in the second stage of being born is so self-evident that it seems almost laughable to refer to it were it not that some authors recommend it in perfect good faith. The use of the forceps is practically contraindicated in these cases: it would be almost certain to end disastrously to the child.

109 WEST EIGHTY-SECOND STREET.

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AN EXPERIMENTAL STUDY OF THE ABSORPTION OF STRYCHNINE IN THE DIFFERENT SECTIONS OF THE ALIMENTARY CANAL OF DOGS.*

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The experimental study of the absorption of strychnine from the alimentary canal which I began some years ago on rabbits[1] I have continued on dogs, because it is an indispensable requirement that results which were obtained from experiments upon one class of animals should be tested in experiments on another class of animals before we think of applying such results to the human being, who is the centre of our interest.

Strychnine is available for the study of absorption chiefly on account of its characteristic reaction upon the animal—the appearance of a tetanus is a sure proof of the absorption of a sufficient quantity. The effective doses and the times which elapse between the administration of the poison and the appearance of the tetanus can serve as units for the comparison of the powers of absorption in the different parts of the alimentary canal in the same class of animals.

In such comparisons, of course, the weight of the animal has to be taken into consideration, inasmuch as it might happen that a large dose will not have any effect in a large animal, while a smaller dose may cause a tetanus in a smaller animal. In the experiments which I am now going to present the effective doses of strychnine were reduced to the unit weight of the animal—*i. e.*, in each case the number of milligrammes of strychnine which proved to be effective were divided by the number of kilogrammes of the weight of the animal, thus establishing the number of milligrammes required for each kilogramme of dog to bring out a pronounced tetanus.

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In each experiment the animal was under ether anæsthesia throughout the entire operation.

The sections of the alimentary canal which were thus separately tested for their absorbent power are as follows: Pharynx, œsophagus, stomach, small intestines, large intestine, and rectum.

The report on these separate sections we shall preface, however, with a few experiments in which the strychnine was introduced into the non-isolated stomach of normal animals, where it is, therefore, uncertain in what section of the canal the absorption in these experiments really takes place.

We are now going to let the experiments speak for themselves.

SERIES A.—*Strychnine in the normal stomach.*

EXPERIMENT 1.—A dog, weighing 22 kg., after having fasted for thirty hours, was slightly narcotized, and 22 mg. of strychnine in 50 c.c. of normal salt solution, colored with indigo-carmine, were introduced through a tube into the stomach. The tube was employed, to avoid the contact of strychnine with the mouth, pharynx, etc., and the liquid was stained, in order to recognize its escape. The animal taken from the table had the first tetanic fit just one hour after the administration of the strychnine; a few more fits followed, but the dog survived. In this experiment 1 mg. of strychnine to 1 kg. of dog brought out a tetanus sixty minutes after administration.

EXPERIMENT 2*a*.—A large dog of 35 kg. was given ether; as in the previous experiment, 20 mg. of strychnine were introduced into the stomach. During observation for three hours there was no effect. Here four-sevenths of a milligramme to the kilogramme of dog had no effect.

EXPERIMENT 2*b*.—The same dog, seven days later. I introduced into the stomach, under the same conditions as before, 30 mg. of strychnine. Tetanus appeared after forty minutes, and death occurred forty minutes later. In this case six-sevenths of a milligramme to the kilogramme brought tetanus after forty minutes.

Some more experiments of the same kind were made, but they will not be reported in detail. All these experiments can be reduced to the following average figures: One and one-sixth of a milligramme of strychnine to one kilogramme of dog brings out a tetanus thirty minutes after the introduction of the poison through a tube into the non-isolated stomach of a normal animal. We might remark here that the usual administration of the poison by the mouth is complicated by the fact that it has to come into contact with the mouth, pharynx, etc., where, as it will be shown later, strychnine is absorbed quite readily.

In presenting the experiments on absorption in the separate sections of the alimentary canal we will commence with the small intestines,

where, in normal conditions, probably the main absorption takes place. By so doing we shall obtain at the same time a unit by which to measure the absorbent power in the other sections.

As far as I am aware no such experiments were yet made by other investigators to determine the absorption of strychnine from the intestines when introduced into them directly.

SERIES B.—*Strychnine introduced into an isolated loop of the small intestines.*

EXPERIMENT 1.—A dog of 13 kg. was given ether, the intestines exposed and tied at a distance of 12 cm. from the pylorus; an opening was made into the stomach and a tube introduced through the pylorus into the intestine; through this tube 40 mg. of strychnine in 10 c.c. normal salt solution stained with methylene-blue were injected; the tube was then withdrawn and the pylorus rapidly tied. Seven minutes after injection, before the sewing-up of the abdomen could be finished, a tetanus broke out, and the animal died eight minutes later.

In this experiment 3 mg. of strychnine to 1 kg. of dog brought out a tetanus after seven minutes.

EXPERIMENT 2.—A dog of 12½ kg. was given ether, the abdomen opened, a loop of the ileum 12 cm. in length tied at both ends, and 15 mg. of strychnine were injected by a hypodermatic syringe into the empty lumen of the intestinal loop. Tetanus broke out in eleven minutes, and the dog died in twenty-one minutes after the injection. Here $1\frac{1}{5}$ mg. of strychnine to 1 kg. of dog brought out a tetanus eleven minutes after the injection.

Here, too, I shall restrict myself to the detailed report of only two illustrative experiments. The average figures are: $1\frac{2}{5}$ mg. of strychnine to 1 kg. of dog introduced into a loop of 12 cm. length of the small intestine will bring out a tetanus in ten minutes.

It is probable that in a longer loop the same effect could be obtained by even a smaller dose. I have, however, restricted my experiments invariably to a loop of the same length.

We turn now to that section of the alimentary canal the absorbent power of which has of late often been the subject of investigation—I mean the stomach. We shall restrict the brief historical remarks to the studies made with strychnine. Here we find that Bouley and Colin[2] maintain that it was absorbed quite rapidly in the stomachs of dogs, cats, etc., when the pylorus was tied. Tappeiner[3] finds that in cats the absorbent power of strychnine was retarded in the stomach when the pylorus was tied. In the investigations of these writers the cardia remained open and the animals often vomited. In my experience on rabbits,[4] whose stomachs were ligated at the cardia and the pylorus, no strychnine was ever absorbed from this organ.

In my present experiments on dogs I have studied the absorption from the entire stomach as well as from the fundus alone.

SERIES C.—*Absorption from the isolated stomach.*

EXPERIMENT 1.—A dog weighing 7300 gr. was given ether, the stomach was exposed and the cardia ligated. An opening was made into the duodenum, and through it 20 mg. of strychnine in normal saline solution, colored with methylene-blue, was injected into the stomach by means of a syringe with a long blunt nozzle; the syringe was withdrawn and the pylorus ligated. For two hours and fifteen minutes the dog was observed, but no tetanus appeared, and not even a sign of hyperæsthesia. The dog vomited often, but only white foam without a tinge of blue, a proof that the ligature of the cardia was tight. To prove that the channels of absorption from the stomach were not cut off by the ligatures around the cardia and pylorus, 8 mg. of strychnine in solution tinged with blue were now injected by means of a hypodermatic syringe into the submucous tissue of the anterior wall of the stomach; six minutes later the animal died in a violent tetanic fit while still under ether.

In this experiment $2\frac{2}{3}$ mg. of strychnine to 1 kg. of dog had no effect upon the animal.

EXPERIMENT 2.—A dog weighing 10 kg. was etherized; the strychnine was introduced through a tube into a normal stomach. The animal had a tetanus after twenty-five minutes, followed by many other fits, but it survived; $1\frac{1}{2}$ mg. of strychnine to 1 kg. of dog introduced into a normal stomach caused tetanus after twenty-five minutes.

The same dog, five days later, was given ether, the stomach was exposed, the cardia ligated, and we injected through the duodenum into the stomach 30 mg. of strychnine in 50 c.cm. colored saline solution, the pylorus being ligated. It was observed for two hours, but no tetanus, and even no hyperæsthesia appeared. Observation had to be discontinued, and the animal was killed with chloroform.

In this experiment it was conclusively demonstrated that while in the normal stomach of the dog $1\frac{1}{2}$ mg. of strychnine to 1 kg. of dog brought out a tetanus twenty-five minutes after administration of the poison into the stomach, but after ligation of the cardia and pylorus 3 mg. to 1 kg. of dog had no effect whatever. This demonstrates distinctly the pronounced retardation of the absorption in the stomach, comparing it with the absorption in the intestines, where 3 mg. of strychnine to 1 kg. of dog would surely bring out a tetanus in seven or eight minutes.

However, the stomach of the dog differs from that of the rabbit, inasmuch as in the dog large doses of strychnine would bring out a

tetanus from the stomach with tied orifices, as can be seen from the following experiments:

EXPERIMENT 3.—Ether was given to a dog weighing 6 kg., the cardia was tied, and I introduced into the stomach through a tube in the pylorus 150 mg. of strychnine in 20 c.c. alcohol (25 per cent.); the pylorus was ligated, the wound sewed up, and the ether discontinued; forty-five minutes later a distinct tetanus broke out, and the animal died after two hours; 25 mg. of strychnine (in alcohol) to 1 kg. of dog brought out a tetanus after forty-five minutes.

I have made a large number of experiments on the absorption from the isolated stomach under varying conditions; either the cardia was first tied and the strychnine was introduced through the pylorus, which was then tied later, or the pylorus was tied first and the strychnine was introduced into the stomach by a tube through the mouth or through an opening in the œsophagus, and then I ligated either the cardia or the œsophagus below the wound. The average figure of all these experiments amount to this: 20 mg. of strychnine to 1 kg. of dog brings out a tetanus forty-two minutes after the introduction of the poison. The smallest dose which was effective in one of these experiments amounted to 9 mg. of strychnine in alcohol to 1 kg. of dog.

The introduction of strychnine into a stomach, the pylorus of which was ligated, but the cardia left open, has not given me clear results, as it was usually complicated by vomiting, which means the bringing of the strychnine into the pharynx, mouth, etc.

I have attempted to study the absorbent power of the fundus of the stomach separately, and the results which I have obtained indicate that the absorptive power is distinctly smaller than that of the entire stomach. This can be seen from the following experiment:

EXPERIMENT 4.—A dog weighing 5 kg. was etherized, the stomach ligated between the fundus and pyloric portion, cardia ligated, a stomach canula tied tightly in the fundus, and through it 100 mg. of strychnine in an alcohol solution introduced into the fundus. The animal was observed for eighty minutes, but no effect, not even hyperæsthesia, was seen. To prove that the paths of absorption were not cut off by the ligatures, 6 mg. of strychnine were injected into the submucous tissue of the wall of the fundus, and six minutes later a violent tetanus appeared. In this experiment even 20 mg. to 1 kg. of dog could bring out no effect.

We should remark here that the absorption which we have seen readily occurs when the drug is introduced into the non-isolated stomach of normal animals, probably takes place in the intestines; and a part of the interval between the administration of the poison and the appearance of the tetanus seems to be consumed by the time required for the poison to pass in sufficient quantity from the stomach into the intestines.

This accounts for the difference in the length of the intervals of the absorption from the normal stomach and from the intestines.

The opinions which we meet with in the older literature on the absorbent power of the œsophagus are simply guesswork and mostly incorrect. The writer of the chapter on absorption, in the *Classical Handbook of Physiology*, by Hermann,[5] proclaims that "while the food is slowly passing through the œsophagus undoubtedly some of it becomes absorbed there." Berg,[6] who described the well-known case of immense dilatation of the œsophagus observed by Luschka,[7] goes so far as to state that the patient's life was supported by absorption of food from the œsophagus. In the case of chronic cardiospasm, with consecutive dilatation of the œsophagus, which I described some years ago,[8] I demonstrated that atropine or alcohol was never absorbed from the œsophagus. In a similar case recently described by Maybaum,[9] it was established that iodide of potash remained unabsorbed in the œsophagus. In the experiments on rabbits I have shown[10] that the œsophagus of these animals absorbs strychnine very poorly. Similar results were obtained in my present experiments on dogs.

SERIES D.—*Absorption of strychnine from the œsophagus.*

EXPERIMENT 1.—A dog of 7 kg. was etherized, the cardia tied, and I injected into the œsophagus 19 mg. of strychnine. The œsophagus was tied above. After one hundred and forty minutes' observation there was no effect, but the animal was found dead the next morning, aspiration pneumonia being the apparent cause of death; $2\frac{5}{7}$ mg. of strychnine to 1 kg. of dog had no effect.

EXPERIMENT 2.—A dog weighing 4 kg., etherized, was similarly treated. I injected into the œsophagus 20 mg. of strychnine, and tied the œsophagus above. After forty-five minutes a weak tetanus appeared. The dog died after two hours. Here 5 mg. of strychnine to 1 kg. of dog brought out a moderate tetanus after forty-five minutes.

The average figures from all the experiments are: $4\frac{1}{2}$ mg. of strychnine to 1 kg. of dog brought out a tetanus after forty-seven minutes.

We should remember that in these experiments the animal, after awakening from the ether, is swallowing very frequently, and that the peristalsis which follows each deglutition passes under the ligature to the lower parts.[11] The strychnine in the œsophagus is therefore very often subjected to a high pressure, which, of course, facilitates the absorption.

Experiments upon absorption within the pharynx cannot be conducted with desirable precision, since this part cannot be well separated from the mouth, nose, and larynx. The data which were derived from our experiments must indeed be interpreted as an expression of the absorption which takes place in all these parts together, though the bulk

of the liquid injected into the pharynx remains there, especially if the animal is kept throughout the experiment on its back, with the head well fixed. Moreover, each experiment was followed up by an autopsy, for the purpose of ascertaining the parts in which the stained solution of strychnine was really located.

SERIES E.—*Strychnine injected into the pharynx.*

EXPERIMENT 1.—A dog weighing 4 kg. was etherized, tracheotomy was performed, and the trachea ligated above; the œsophagus was tied just below the pharynx. I introduced into the pharynx 12 mg. of strychnine in 5 c.c. of a stained solution. Tetanus followed after twenty-five minutes, and death one hour later. Autopsy showed blue saliva mainly in the pharynx, but also in the mouth and in the larynx. In this experiment 3 mg. of strychnine to 1 kg. of dog brought out a tetanus within twenty-five minutes. This can also be taken as an average figure from all the experiments in which tracheotomy was performed. In experiments without tracheotomy the tetanus appeared much earlier after injection and with smaller doses. The autopsies have shown, however, that the blue stain extended deep into the trachea. This observation gave rise to the following experiment:

A dog of 11½ kg. was etherized, and I injected directly into the trachea 15 mg. of strychnine. Tetanus occurred after six minutes. There were fits, with very short intermissions, until death set in ten minutes later. Here 1½ mg. of strychnine to 1 kg. of dog brought out strong tetanic attacks in six minutes.

The absorbent power of the large intestine does not seem to differ materially from that of the small intestines, but the presence of some fecal masses within the colon seems to retard the absorption somewhat, as can be seen in the following experiments:

SERIES F.—*Absorption in the colon.*

EXPERIMENT 1.—To a dog weighing 12½ kg. ether was administered, and the colon, which was not entirely empty, was tied near its junction with the small intestine. An opening was made in the lower part, and 20 mg. of strychnine in a 6 c.c. saline solution injected into the upper part, and the bowel tied immediately. The length of the isolated gut was about 12 cm. The first tetanic fit occurred after twenty-three minutes, the animal dying one hour later. In this experiment 1⅝ mg. of strychnine to 1 kg. of dog produced a tetanus in twenty-three minutes.

EXPERIMENT 2.—A dog of 7½ kg. was given ether and the colon exposed. It was empty. A piece 12 cm. in length was isolated by ligatures, and 10 mg. of strychnine was injected into the lumen of this piece by means of a hypodermatic syringe, and the puncture clamped. The first tetanus appeared nine minutes after the injection, and the

animal died after twenty-three minutes. Here $1\frac{1}{3}$ mg. of strychnine to 1 kg. of dog brought out a tetanus after nine minutes.

There have been, however, some variations with regard to doses and time of absorption even in experiments with a perfectly empty colon.

The average figures of these experiments are: $1\frac{1}{5}$ mg. of strychnine to 1 kg. of dog brings out a tetanus in fourteen minutes.

In the experiments upon the rectum we have studied the effect of the injection of strychnine in a non-isolated normal rectum as well as in a rectum which was separated from a colon by means of a ligature. In the first case we are not studying the absorption in the rectum alone, as a part of the injected liquid is surely carried up into the colon or perhaps even into the small intestines.

It deserves to be mentioned especially that in the experiments on the rectum this organ was rarely empty.

On account of the practical importance of these experiments we will describe here in detail a greater number of them.

SERIES G.—*Experiments upon the rectum.*

(*a*) Normal rectum.

EXPERIMENT 1.—Dog of 17 kg.; I injected into the normal rectum 18 mg. of strychnine; after fifteen minutes a tetanic attack broke out; a few more fits followed, but the animal survived. In this experiment $1\frac{1}{17}$ mg. of strychnine to 1 kg. of dog brought out a tetanus after fifteen minutes.

EXPERIMENT 2.—Dog of 22 kg.; I injected 22 mg. of strychnine into the normal rectum. Tetanus occurred after fifteen minutes, and the animal died after an hour. Here again 1 mg. of strychnine to 1 kg. of dog brings out a tetanus after fifteen minutes.

EXPERIMENT 3.—A dog of 23 kg., which received 300 mg. of strychnine in the isolated stomach, was observed for over two hours, but no effect appeared. Now 50 mg. of strychnine was injected through the anus into the rectum; after nine minutes a violent tetanus appeared, and the animal died ten minutes later.

(*b*) Rectum tied about 15 cm. above the anus.

EXPERIMENT 4.—A dog of $11\frac{1}{2}$ kg. was etherized, the rectum tied, and I injected through the anus 20 mg. of strychnine in 6 c.c. of water, colored with methylene-blue; at least one-half of the liquid came out again. Tetanus appeared after fifteen minutes, and death after half an hour. Here about $\frac{4}{5}$ mg. of strychnine to 1 kg. of dog brought a tetanus in fifteen minutes.

EXPERIMENT 5.—Dog of $8\frac{1}{2}$ kg.; after etherization the rectum was tied, and I injected 10 mg. of strychnine into the lumen through the wall of the gut by means of a hypodermatic syringe; the puncture was clamped. After eight minutes a strong tetanus appeared; the animal

died after twenty-five minutes. In this experiment 1½ mg. of strychnine brought out a tetanic fit in eight minutes.

EXPERIMENT 6.—A dog of 7 kg. was similarly injected with 20 mg. of strychnine, and the puncture clamped. A violent tetanus broke out after four minutes, and the animal died six minutes later. Here 2⅞ mg. of strychnine brought out a tetanus in four minutes.

EXPERIMENT 7.—A dog of 5700 gr. was etherized, the rectum tied, and I injected through the anus 20 mg. of strychnine in 6 c.c. of colored water; a few drops were expelled. A violent tetanus occurred in two minutes, and death one minute later. In this experiment 3½ mg. of strychnine to 1 kg. of dog gave a tetanus in two minutes.

The average figures from these and other experiments upon the isolated rectum are: 1⅘ mg. of strychnine to 1 kg. of dog brings a tetanus in seven minutes.

In the following three tables a condensed presentation is to be found of the results of the experiments upon the different sections of the alimentary canal. The first table gives that experiment upon each section in which the minimum effectual dose was used. The second table gives the experiment with the shortest interval. The third table gives *average figures* for each series of experiments, which were obtained by the following simple method: The amount in milligrammes of strychnine to the kilogramme of dog, which were employed in every experiment, were added together, and the sum total was divided by the number of experiments made in this series. The same was done with the intervals between the injection and the appearance of the tetanus. All the intervals, in minutes, were added together, and the sum-total was divided by the number of experiments in this series. Both numbers, which were thus composed from all the experiments in each series, were linked together to an average figure which might present at least in an approximate way the absorbent power for strychnine of the corresponding section.

It should be remembered that in starting out to study the absorbent power by means of the characteristic effect of strychnine we had two criteria in mind, by means of which the degree of absorption could be estimated. One of these was the dose which brings out a tetanus, and the other was the time which passes between the administration of the poison and the appearance of the tetanus. Both criteria, the dose and the time, have in common that they are in inverse proportion to the power of absorption—*i. e.*, absorption is increased when they are decreased—in other words, the smaller the dose which can bring on a tetanus, and the shorter the interval between the administration of strychnine and the outbreak of the fit, the greater is the power of absorption of the part in which the strychnine was introduced. But both criteria are in inverse proportion to each other, and cannot be observed

during one and the same experiment. The smaller the dose which can effect a tetanus the longer is the interval between the appearance of the tetanus and the administration of strychnine, and, on the other hand, in order to obtain a shorter interval a larger dose has to be employed. However, by compounding average figures from the entire series of experiments with the minimum dose, as well as those with the shorter intervals, we obtain a measure for the absorbent power in which both criteria are equally represented. We can now state that the first table presents the power of absorption as adjudged by the criterion of the dose, the second table that judged by the criterion of the interval, and the third table shows the absorbent power as compounded from both criteria together.

TABLE I.—SHOWS THE EXPERIMENTS WITH THE EFFECTIVE MINIMUM DOSES OF STRYCHNINE IN MILLIGRAMMES REDUCED TO 1 KILO OF DOG.

Section of alimentary canal.	*Strychnine.*	*Interval between injection and tetanus.*
Normal stomach	$^6/_7$ mgr.	40 minutes.
Small intestines	1 "	13 "
Isolated stomach	9 "	20 "
Œsophagus	4 "	50 "
Pharynx	$1^3/_5$ "	20 "
Colon	$1^1/_5$ "	9 "
Rectum	$^4/_5$ "	15 "

TABLE II.—SHOWS THE EXPERIMENTS WITH SHORTEST INTERVAL BETWEEN INJECTIONS AND APPEARANCE OF TETANUS.

Section of alimentary canal.	*Strychnine.*	*Interval between injection and tetanus.*
Normal stomach	$1^1/_2$ mgr.	25 minutes.
Small intestines	$2^1/_2$ "	8 "
Isolated stomach	23 "	15 "
Œsophagus	5 "	45 "
Pharynx	$1^3/_5$ "	20 "
Colon	$1^1/_5$ "	8 "
Rectum	$3^1/_2$ "	2 "

TABLE III.—AVERAGE FIGURES.

Section of alimentary canal.	*Strychnine.*	*Interval between injection and tetanus.*
Normal stomach	$1^1/_6$ mgr.	31 minutes.
Small intestines	$1^2/_5$ "	10 "
Isolated stomach	20 "	42 "
Œsophagus	$4^1/_2$ "	47 "
Pharynx	3 "	25 "
Colon	$1^4/_5$ "	14 "
Rectum	$1^4/_5$ "	7 "

I believe that the experiments embodied in this paper have established the following points:

The absorption of strychnine in the stomach is incomparably lower than in any other section of the alimentary canal, and, furthermore,

the absorbent power of the fundus seems to be even lower than that of the entire stomach.

The power of absorption in the œsophagus is somewhat better than in the stomach, but is still considerably inferior to the absorption within the other parts of the alimentary canal.

The power of absorption in the other three parts of the canal—*i. e.*, the small intestines, colon, and rectum—seems to be equal. The differences which were observed are too small and the method not exact enough to justify the assumption of an actual difference in the absorbing power of these parts. However, these experiments brought out the following remarkable facts: that the isolated rectum absorbs at least as well as the small intestines; that in some experiments the effects appeared earlier when the rectum was isolated than when it had free connection with the balance of the gut; that from no part of the canal were such short intervals between the injection of the strychnine and the appearance of the tetanus observed as were frequently seen when it was injected into the rectum—in the latter case the interval was sometimes only two or three minutes; finally, impaction of the rectum proved to be no hinderance to the prompt absorption of the poison therefrom.

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A CRITICAL SUMMARY OF THE LITERATURE

ON

SPLENIC PSEUDOLEUKÆMIA (ANÆMIA SPLENICA; SPLENOMEGALIE PRIMITIVE).[1]

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HISTORY OF THE DISEASE. The history of splenic pseudoleukæmia is inseparably connected with that of the diseases to which it is closely allied—namely, leukæmia and the other forms of pseudoleukæmia. Its

[1] For the author's report of a case, see THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, October, 1899, vol. cxviii., No. 330, p. 428.

study, therefore, may well trace the development of our knowledge of those diseases as a class. The attention of the medical profession was early directed to a complexus of symptoms characterized by enlargement of the spleen, pallor, hemorrhages, œdema, and grave cachexia. Hippocrates wrote on the association of splenic tumor with dysentery, hemorrhage, scorbutus, and miasmatic diseases. Galen described hypertrophy of the spleen in protracted autumnal fever and other cachectic states. Malpighi, Morgagni and others observed splenic and lymphatic enlargement associated with pallor, hemorrhage, and emaciation, which progressed to a fatal termination. Nivet[1] mentions scorbutus, rhachitis, and scrofula among the many causes of splenic enlargement. In some of the cases of splenic hypertrophy reported by him under the heading of rhachitis and scrofula, extensive glandular enlargement and grave cachexia developed during the course of the disorders. There is much in the literature of the older writers to indicate that they were quite familiar with hypertrophy of the spleen in miasmatic disease, and that they also had noted the condition in various other affections, such as scrofula, continued fevers, syphilis, and liver disease. Now and then one also finds record of cases in which, without apparent cause, the spleen enlarged, hemorrhages and cachexia supervened, and death followed. No particular types of disease of this nature were well recognized, however, until, in 1832, Hodgkin wrote his now well-known article on "Some Morbid Appearances of the Absorbent Glands and Spleen."[2] He reported a number of cases which showed an increase in the size of the lymph-glands and spleen, followed by emaciation, ascites, pallor, œdema, and death. The blood was not examined in any of the cases, and the report in many instances is meagre. One is able to discern, however, that undoubtedly the author's cases included carcinoma, tuberculosis, sepsis, and probably other morbid conditions. Nevertheless, it was the first worthy attempt to group this class of affections. In October, 1845, Bennet published the report of a case of hypertrophy of the spleen in which the blood of the patient contained a great number of white corpuscles resembling pus-cells. In November of the same year Virchow reported a similar case, and correctly attributed the large increase in the white cells to an augmentation of the white blood-corpuscles. He believed that the great increase in the leucocytes was due to the hypertrophy of the spleen. Subsequently, he evolved the doctrine of the disease, leukæmia, which he described as an affection characterized by cachexia, enlargement of the spleen and lymph-glands, and an augmentation in the number of white blood-corpuscles. Other histological changes of the disease described by him were cellular hyperplasia of the lymph-glands and spleen and infiltration of lymph-cells

[1] Archives Generales de Medicine, 1838. [2] Medico-Chirurgical Transactions.

in the tissue of the liver, lungs, kidneys, and intestinal tract. Virchow recognized a lymphatic and a splenic form of leukæmia. In the year 1868 the work of Neumann and Bizzozero, on the hæmatopoietic function of the medulla of bone, gave rise to a new field of clinical observation, and soon a sufficient number of cases of leukæmia was recognized in which profound changes in the medullary substance of the bone were present to warrant a third subdivision of the disease—namely, myelogenic leukæmia. Shortly after Virchow's description of leukæmia, Bonfils, Trousseau, Wunderlich, Wilkes and others observed cases which presented extraordinary swelling of lymph-glands and spleen and many of the clinical features of leukæmia. It was noted, however, that the number of white blood-corpuscles was not increased. In order to distinguish these cases from leukæmia, Trousseau proposed the name *adenia*, by which the French still recognize the disease. Wilkes added that of *anæmia lymphatica*, or *Hodgkin's disease*. Wunderlich described it as *progressive glandular hypertrophy;* Virchow, as *lymphosarcoma;* Billroth, as *malignant lymphoma*, and Cohnheim, as *pseudoleukæmia*. Aside from the marked enlargement of the lymph-glands and spleen, the disease was characterized by pallor, hydrops, and a tendency to hemorrhages, cachexia, final marasmus, and death. The cases did not include similar clinical manifestations due to malaria, syphilis or any other apparent cause. The essential feature which distinguished the affection from leukæmia was the absence of an increase in the leucocytes. In many cases the lymph-glands only were enlarged; in other cases both the spleen and lymph-glands showed marked increase in size. To the first class of cases the older writers applied the terms *adenia, anæmia,* and *pseudoleukæmia lymphatica*. To the second class were applied the terms *adenia, anæmia,* and *pseudoleukæmia splenolymphatica*. More recently a relatively small number of cases have been reported, belonging to this class of diseases, in which the lymph-glands were unaffected, while the spleen was greatly hypertrophied. These cases present a most striking analogy to splenic leukæmia, the essential difference being in the alterations in the blood. The number of cases thus far recognized clinically does not exceed twenty-five, and the number of autopsies is still much smaller. The uniformity of the clinical and pathological features of the disease, however, establish the affection as a distinct morbid entity, which is, in reality, a splenic form of pseudoleukæmia. In 1856, Woillez described the first undoubted case of this disease under the title "Observations d'hyp. de la rate" (*Union Medicale*). His report referred to a man, forty years of age, who was taken sick without apparent cause. The disease was characterized by anæmia and a rapidly progressive course, which terminated in death. There was no increase in the number of leucocytes; the spleen measured 26 x 12 cm. Two enlarged glands were found in the

mesocolon. The first case recognized as belonging to this type of disease was reported by Gretzel, from Griessenger's clinic, under the name of anæmia splenica.[1] Since the term anæmia splenica, by which the disease under consideration has been most frequently reported, originated with this case, I feel justified in giving its details: A child, ten months old, pale and weak from birth, with tendency to cough and diarrhœa, presented a moderate enlargement of the liver and a great enlargement of the spleen. The lymph-glands were palpable. The symptoms gradually increased in intensity until, at the end of six months, the child died. Multiple enlargement of lymph-glands was present everywhere. There was no leucocytosis. Autopsy by Cohnheim showed hyperplasia of normal elements of spleen, with incipient cirrhosis of the liver and lymph-glands. Griessenger recognized that the case differed from leukæmia solely in the absence of a leucocytosis. He, among others, believed that such cases should be given a distinguishing name. Cohnheim[2] had already applied the term pseudoleukæmia to such affections; but Griessenger had accepted the term anæmia lymphatica, proposed by Wilkes, as applicable to those cases in which the lymph-glands were chiefly involved. It was, therefore, natural for him to apply the term anæmia splenica to similar cases in which the splenic enlargement predominated. In reality, the case described from Griessenger's clinic belongs to that pathological state described more recently by v. Jaksch as anæmia infantum pseudoleucæmica. Müller reported seven cases of pseudoleukæmia.[3] Among the number was one which undoubtedly belongs to the form of splenic pseudoleukæmia. Autopsy showed moderate enlargement of the liver and marked enlargement of the spleen. The splenic hypertrophy was due to an increase in its normal elements. Under the name of anæmia splenica, Strumpell[4] describes a case which has been quoted by nearly all who have written on the subject since that time. Banti claims that this was undoubtedly a case of progressive pernicious anæmia. It certainly does not correspond to splenic pseudoleukæmia. To Banti[5] must be given the credit of first describing the affection in such a manner as to bring the subject clearly before the medical profession. Unfortunately, his publication was in Italian, which undoubtedly restricted its influence. He reported three cases, including two autopsies, from his own observations, and collected cases, previously reported, which he thought belonged to this class. He chose to perpetuate the term which Griessenger had applied, and described the affection as "anæmia splenica." Since then cases presenting the same clinical manifestations

[1] Berlin. klin. Wochenschrift, 1886.
[2] Virchow's Archives, Bd. xxxiii.
[3] "Uber idiopath. Milztumoren," Berlin. klin. Wochenschrift, 1867.
[4] Archiv der Heilkunde, 1875.
[5] "Dell' Anæmia Splenica," Archivo della Scuola d' anat. pathologica, 1888.

and pathological features have been described under various names. Debove and Bruhl applied the term splenomegalie primitive; Eichhorst, splenic pseudoleukæmia; Gaucher, idiopathic hypertrophy of the spleen without leucocythæmia. (For reports of other cases, see references.) The manifold nomenclature arising in part from the different theories held by the different authors regarding the nature of the disease gives rise to a certain amount of confusion concerning the affection. At best, the terms leukæmia, pseudoleukæmia, and splenic anæmia simply designate a certain grouping of clinical manifestations and pathological changes.

If the ordinary forms of Hodgkin's disease were described as anæmia lymphatica and anæmia splenolymphatica it would be consistent to apply the term anæmia splenica to the rare cases under consideration. However, since the term pseudoleukæmia is more often applied to such conditions, I believe the appropriate name for this form of the disease is splenic pseudoleukæmia.

Etiology. The etiology of splenic pseudoleukæmia is unknown. The disease does not appear to bear any direct relation to malaria, syphilis, tuberculosis, or alcoholism. It seems to arise independent of all constitutional disorders. The cultures obtained from the spleen in my case, both ante-mortem and post-mortem, contained no organism that could be regarded as specific, and the spleens of animals injected with blood from the spleen of the patient showed no changes. The microscopical examination of the spleen and other organs revealed no micro-organisms. In 1894, Sciolla obtained negative results from cultures made from the spleen ante-mortem. The negative results in the two cases mentioned do not, of course, exclude micro-organisms as a cause of the disease. However, certain features in connection with the disorder render it very improbable that a specific micro-organism is the etiological factor. The disease is extremely rare, and has never occurred endemically or epidemically. Isolated cases have appeared in different parts of the world. More cases have been recognized in Italy than in any other section. There appears to be no special predisposing cause of the disease.

Pathological Anatomy. I find record of only about ten post-mortems in which it is reasonably certain that splenic pseudoleukæmia was the cause of death. As a rule, the cadaver presents a high grade of emaciation. In some cases the subcutaneous fat has been well retained. The skin and conjunctivæ are subicteric, and punctiform hemorrhages may be visible in different parts of the body. Usually the subcutaneous connective tissue of the dependent parts is infiltrated with serum, and œdema of the extremities is often present. Visible external glandular enlargement is seldom found, yet it is not rare to be able to palpate a few glands, the size of a bean or a hazel-nut, in the inguinal or axillary

regions. The abdomen usually contains a variable quantity of ascitic fluid. Ecchymoses may be present beneath all the serous surfaces. The pericardial sac often contains a serous fluid. The heart is generally slightly dilated, and frequently presents signs of granular and fatty degeneration of its muscle, such as is usually found in severe anæmia. The pleural cavities often contain fluid, and when the spleen is extremely large, compression of the left lung may give rise to atalectasis of its lower lobe. In a few cases a real pleurisy has been present on the left side. Although nausea, vomiting, and diarrhœa may be prominent symptoms throughout the course of the disease, the gastro-intestinal tract does not present serious alterations. The glands at the base of the tongue have been found somewhat enlarged. Not infrequently Peyer's patches and the solitary follicles show slight hyperplasia. The liver is usually enlarged. In my case it weighed 2800 grammes, which exceeds by far the usual weight recorded in this affection. Its surface is smooth, the edges round, and the organ presents, as a rule, no visible signs of cirrhosis. On section, the organ is uniform in firmness, cuts easily, and its consistency is not apparently increased. Histology of the liver: The liver shows slight increase in its interlobular connective tissue. Infiltration of round cells is observed about the portal vessels and between the liver lobules. In a case reported by Banti there was considerable increase in the connective tissue, which he described as an interstitial perilobular hepatitis. The liver cells may be normal. Here and there, where there is considerable proliferation of connective tissue, the hepatic cells may be atrophic. The spleen is always increased in size, and often fills the greater part of the left half of the abdominal cavity. The normal size of the spleen is 12 x 8 x 3 cm.; its normal weight is 200 grammes. In my case it measured 31 x 16 x 9.5 cm. and weighed 2350 grammes. The normal shape and outline of the organ is preserved. Its surface is usually smooth, although in some cases areas of peritoneal thickening resulting from perisplenitis are present. Rarely peritoneal adhesions have been recorded. The organ is firm. In a case reported by Banti there was a visible increase in the connective tissue. Small white areas, about the size of a pea, were noted by him, which proved to be made up of increased connective tissue. In my case there was no macroscopic increase in the connective tissue. The Malpighian corpuscles were not visible. Histology: The Malpighian corpuscles are usually not markedly altered. Banti records considerable sclerosis of these bodies in one of his cases. He summarized his findings in the spleen as (1) atrophy and sclerosis of the Malpighian corpuscles; (2) the substitution of an irregular and fibrous reticulum for the normal fine reticular substance of the splenic pulp. The following quotation is from a case reported by West: "The spleen showed slight increase in the trabecular tissue—*i. e.*, slight fibrosis. Malpighian corpuscles are

much diminished in size and badly developed. Iron reaction negative." For the changes in the spleen in my case the reader is referred to the histology given in the report. The kidneys are anæmic. The epithelium of the tubules may show signs of granulo-fatty degeneration. A real nephritis, however, is not present. The pancreas and adrenals are negative. The lymphatic glands are usually not enlarged. In some cases, however, isolated glands, or small groups of glands, especially in the retroperitoneal region, have shown slight enlargement. They are grayish and somewhat red, and firm in consistency. On section they show no softening, and the microscope reveals hyperplasia of both the principal elements. The mesenteric glands may show similar changes. The medulla of the bone often presents an appearance closely resembling that found in primary pernicious anæmia. In one of Banti's cases he notes that the medulla in the long bones was, as a rule, fatty; but that in both femora a fœtal condition of the marrow was present. In my case the compact tissue of all the bones was very much reduced, producing a considerable degree of brittleness. The sternum could easily be broken with the fingers. Throughout the length of the femur, tibia, and humerus the normal fat was replaced by dark red marrow. Indeed, red atrophy of the marrow was distinctly present in every bone examined, including, beside those mentioned, the ribs, clavicle, skull, vertebræ, and small bones of the feet. The blood changes found in the disease are described under the symptomatology.

Symptomatology. The following description of symptoms is based on those present in my case, supplemented by a study of the cases most carefully reported. The manner of onset varies; as a rule, however, the disease begins very insidiously, so that it is often difficult to establish the period at which the first manifestations of the affection appeared. In many cases the first symptoms noted are cardiac palpitation on exertion, accompanied by a feeling of unusual muscular fatigue. Headache and slight pallor of the mucous membranes may be present. Early in the disease the appetite and digestion are usually good and the bowels regular. After a period varying from a few weeks to two or three months the attention of the patient is attracted to the existence of a tumor in the left hypochondriac region. The tumor is seldom painful, and disturbs the patient chiefly by a sense of weight and tension in that region. In some cases, as in my own, a tumor in the left hypochondriac region is the first symptom noted by the patient. Shortly after this discovery, owing in part, no doubt, to the mental concern of the patient in regard to his disorder, a feeling of weakness on muscular exertion is experienced. Following this may appear nausea and vomiting and at times persistent diarrhœa. Dyspnœa and cardiac palpitation on exertion may be rather prominent features. Now and then dizziness and headache may be experienced. Epistaxis is a common early symptom.

In other cases a tumor may appear in the left hypochondriac region, which may increase in size for nearly a year without any disturbance in the general health or even local disturbance, aside from a feeling of weight or tension in the part. After a variable time—seldom longer than a year—the symptoms of anæmia and general disorder in organic function supervene. In rare cases, as in one reported by Debove, the local phenomena referable to the spleen may be very distressing. Pain, paroxysmal in character, simulating hepatic colic, with attacks of nausea and vomiting, may accompany the gradual loss of muscular power and signs of anæmia. Examination of the patient may then reveal the signs of marked anæmia and a palpable tumor in the left hypochondriac region. It matters not what the mode of onset is; after the disease has developed the clinical picture will present the same essential features: (1) Hypertrophy of the spleen; (2) absence of marked lymphatic enlargement; (3) anæmia progressing toward a cachectic state. At this time the symptoms are all very pronounced, with the exception of pain, which may be only slight or entirely absent throughout the whole course of the affection. Debility is great; œdema appears, at first transitory in character, involving the ankles and wrists. A vespertine fever, preceded by chilly sensations, may be present. Gastro-intestinal disturbances, manifested in anorexia, indigestion, and diarrhœa, are present in varying degrees of intensity. Finally, within a period of a few weeks or months, the cachexia of anæmia becomes pronounced. Emaciation gradually increases. The skin and conjunctivæ become subicteric; pallor peculiar to profound anæmia appears. Muscular weakness confines the patient to his room or bed. Œdema becomes permanent and is associated with ascites and hydrothorax. Petechial hemorrhages on the extremities and in the mucous membranes evidence the high grade of malnutrition of the capillary walls. Persistent and often incoercible diarrhœa exhausts the patient, and involuntary evacuation of the bowels may announce the approach of dissolution. At the beginning of the cachectic stage the blood examination usually shows a grade of anæmia which approximates very closely the findings in chlorosis. As the cachexia increases the number of red blood-corpuscles, as a rule, diminishes, until finally, as in my case, less than one million per cubic millimetre may be registered, with hæmoglobin so slight in amount as to preclude the possibility of accurate estimation. At no time, however, is there a leucocytosis presenting the characteristics found in leukæmia. From a scientific as well as therapeutic stand-point it is extremely interesting to note that so far as may be gleaned from the histories of the cases, the splenic hypertrophy has preceded or at least has developed contemporaneously with the appearance of the anæmia. In no case belonging properly to this type of disease, so far as I have been able to discern, has the splenic hypertrophy followed the appearance of the anæmia. In the majority

of cases the patient has not consulted the physician until his attention has been attracted to the tumor in the side. In my case the patient, naturally a quick observer, was not conscious of any unusual physical manifestation until he discovered a tumor a few inches below the costal arch in the left hypochondriac region. The fact that splenic hypertrophy is apparently the primary condition has an important bearing on the treatment of the disease. It also contributes much to the nature of this type as well as the other forms of pseudoleukæmia. As a rule, in the progress of the disease the size of the spleen bears a direct relation to the grade of anæmia. However, there are exceptions, in which the spleen is enormously enlarged before anæmia is pronounced. In still other cases anæmia may be profound when the splenic tumor is of moderate size. After cachexia develops the disease is usually rather rapidly progressive, and death is due to complications, or it follows a period of extreme marasmus. The disease may be divided into three stages. The first stage includes an indefinite period, during which the spleen begins to enlarge. The second stage may conveniently be said to include that period during which the symptoms of anæmia develop, accompanied, as a rule, by gastro-intestinal disturbances and general asthenia. The third stage is that of cachexia. This stage is usually ushered in with œdema, followed by a somewhat regularly progressive aggravation of all the symptoms of the previous stages, and finally terminates in extreme marasmus and death.

Special Symptoms. *Splenic Hypertrophy.* So far as is known, all the disorders of the spleen that are of clinical importance are accompanied by its enlargement. Splenic pseudoleukæmia presents as one of its essential features an enlargement of the spleen. The enlargement is uniform, constituting a true hypertrophy in a clinical sense. Its size varies with the stage of development of the disease, and is not the same in all cases. The spleen enlarges gradually, and is not subject to marked fluctuations in size. Owing to the fact that the first stage of splenic enlargement is seldom observed, it is impossible to estimate its rapidity of growth from the beginning. In my case, after the spleen was first palpable, it gradually enlarged until its lower border reached a point two fingers'-breadth above the pubic bone, and its inner border extended beyond the median line, the patient in the upright position. In this case it appears that the enlargement was exceptionally great. Usually the organ extends no further than one or two hands'-breadth below the costal arch. Its outline is symmetrical. As a rule, no irregularities are to be felt, either on its margin or surface. Its consistency is firm. The splenic notch occupies its usual position. The organ is respiratory, movable in a diagonal direction, peculiar to splenic enlargement, and permits of lateral movement. Pain on pressure may or may not be present. In my case a loud souffle was to be heard all over the

enlarged organ. In some cases friction rubs, due to perisplenitis, have been heard. A perisplenitis may result in local thickenings that may be felt as irregularities on the surface of the organ. When the enlargement is marked the left half of the abdomen is more prominent than the right, and the region of the left costal arch appears bulging. In my case, just previous to death and at the time of great diarrhœa and involuntary discharges, the spleen was reduced fully two inches in its various diameters. The enlargement of the spleen may be unaccompanied by pain. In the majority of cases an uncomfortable sensation of weight and fulness in the left hypochondriac region leads to the discovery by the patient of the tumor. In rare cases neuralgic pains or paroxysms resembling hepatic colic, accompanied by obstinate vomiting, appear as early manifestations of the disease. At times the pain may be so intense as to demand radical means for its relief. The explanation of the pain is not always apparent. A perisplenitis is often found, but pain has been a marked feature when such a condition was not present. In all cases when pain exists it is increased by pressure.

Lymph-glands. The lymph-glands may remain normal in size throughout the course of the disease. It is not uncommon, however, for the post-mortem to reveal enlargement of the retroperitoneal and bronchial glands, either isolated or in groups. The glandular hypertrophy, if present, is always moderate. In a few cases, after splenic hypertrophy had existed for a long time and the symptoms of the disease were well developed, small kernels have appeared in the axillary and inguinal regions. The glands at the base of the tongue and the tonsils have been found enlarged.

Liver. The liver is nearly always considerably enlarged; its surface and outline are regular, its consistency firm, and its lower border sharp. Perihepatitis may occur.

Anæmia. Anæmia is one of the characteristic features of the disease. So far it has not been recorded in any case that the signs of anæmia preceded the development of splenic hypertrophy. The symptoms of anæmia develop gradually during the second stage of the affection, until that state of profound alteration of general nutrition is reached which is clinically designated by the term cachexia. During this stage dizziness, tendency to syncope, headache, dyspnœa on exertion, and cardiac palpitation are present in variable degrees of intensity. Pallor of the mucous membranes gradually develops. Hæmic heart-murmurs may be heard. The skin assumes a hue peculiar to anæmia—pallid at first and later on of a yellowish tinge. The conjunctivæ become slightly subicteric. Epistaxis, such as may occur in anæmia from any cause, is often a frequent and early symptom. The hemorrhage is seldom profuse. The advent of cachexia is characterized by a tendency to œdema and effusion into serous cavities, marked asthenia, hemorrhages, fever, and an aggravation of all the pre-existing symptoms. The mucous mem-

branes become exceedingly pale. The carotids pulsate visibly. Capillary pulse may be present. A venous hum is often noted. The patient gradually emaciates. Persistent diarrhœa often supervenes in the later stages, and anæmia progresses to the highest degree. A period of extreme marasmus finally terminates in death.

Cachexia. The cachexia of splenic pseudoleukæmia does not differ from the cachexia of carcinoma, tuberculosis, or any other profound disturbance of nutrition. Although epistaxis is frequently an early symptom, hemorrhage from other organs than the nose seldom takes place until cachexia develops. During this stage hæmatemesis, hæmoptysis, melænæ, and hæmaturia have been observed, and petechiæ of the skin and mucous membranes is common. Retinal hemorrhages are to be expected.

Fever. Fever may be present during the early stage of the affection. It is a variable symptom, and may be absent for weeks at a time, and then irregular pyrexia may develop. It may be due to a localized peritonitis. Pyrexia independent of local causes is seldom pronounced until the stage of cachexia, when from time to time an afternoon temperature of 102° may be registered. The fever is similar to that which is present in leukæmia and all grave anæmiæ. No satisfactory explanation has ever been given for its occurrence.

Circulatory Symptoms. The heart may be slightly dilated; hæmic murmurs are often heard, especially in the later stages. The carotids and other large arteries throb visibly, and a capillary pulse is not infrequently noted. The pulse is full and frequent, and may approach the water-hammer character. The veins are often prominent, and the bruit de diable may be heard in the neck.

Pulmonary Symptoms. Râles due to compression of the lung by the splenic tumor are often present at the base of the left lung. Dyspnœa, due to deficient hæmoglobin and the elevation of the diaphragm, is experienced.

Digestion. As a rule, the appetite is fair until the disease is well advanced, when it gradually fails. Nausea and vomiting may be early symptoms, and when associated with paroxysms of pain in the region of the spleen may cause much distress. The gastric disturbances may be due to pressure, or, possibly, may be reflex through involvement of the sympathetic nervous system. Diarrhœa may be an early symptom and persist for several weeks or months. Constipation often alternates with diarrhœa. Toward the end diarrhœa may be incoercible, and at last involuntary evacuations may take place. Hemorrhage from the stomach has been recorded.

Nervous System. The symptoms referable to the nervous system are those chiefly of anæmia—namely, vertigo, headache, neuralgia, and mental fatigue. The mind usually remains clear until near the end.

Blood. The blood varies with the stage of the disease. In the begin-

ning of the affection there is only a slight reduction in the number of red blood-corpuscles and a relatively greater diminution in the hæmoglobin. After the disease is well advanced the blood is very pale and thin. Its coagulability is very much reduced. Red blood-corpuscles: The red blood-corpuscles are, as a rule, very much diminished in number. In the few cases in which they have been counted the results have ranged from about 4,000,000 to 568,000 per c.mm.; the average case will show about 2,500,000 per c.mm. at the beginning of the stage of cachexia, and as the disease progresses the number usually decreases. There is no marked alteration in the form and size of the corpuscles. Poikilocytosis has not been recorded. The corpuscles are pale. Nucleated red blood-corpuscles have not been observed. Roleaux formation is absent in the later stages. The hæmoglobin is reduced in quantity; the individual corpuscle contains less hæmoglobin than normal. The color index, however, does not seem to be as low as in chlorosis. In my case, although the hæmoglobin was reduced to 10 per cent., the color index was never lower than about 0.85. White blood-corpuscles: In the majority of cases the leucocytes are normal in number, and the relation of the different forms is not disturbed. No abnormal elements have been recorded. In one of Banti's cases the number was increased to 19,805 per c.mm., but in this case pneumonia was present as a complication. In Franzolini's case five times the normal number were found. Under such circumstances the character of the leucocytes alone would enable one to differentiate the disease from splenic leukæmia.

Urine. The urine is normal in quantity and may not show special changes. A large quantity of nucleohistone was found by Stein in a clinical case of splenic pseudoleukæmia. The solids of the urine are not altered in a characteristic manner. A trace of albumin has been found. Signs of extensive degenerative changes in the kidney have not been observed; sugar is absent. I was able to produce alimentary glycosuria by administering a moderate quantity of glucose. This was due, no doubt, to impairment of the function of the liver.

DIAGNOSIS. Essential to the diagnosis of splenic pseudoleukæmia is (1) an idiopathic splenic hypertrophy; (2) a progressive anæmia without a leucocytosis presenting the characteristics found in leukæmia; (3) the absence of marked lymphatic enlargement. Since neither signs of anæmia nor local sensations have been noted previous to the development of splenic hypertrophy, the disease could scarcely be suspected before the spleen was palpable. In relation to splenic hypertrophy, one must determine whether the palpable structure in the left hypochondriac region is a splenic tumor, and, if so, its nature. As a rule, tumors of the spleen present less difficulty in diagnosis than those of any other abdominal structure. They emerge from beneath the left costal arch and cling closely to the abdominal wall, overriding all structures in the

abdominal cavity. Their development is downward, along a line which is directed toward the right pubic bone.

Splenic enlargements are respiratory movable, and in a diagonal direction, which is peculiar to splenic tumors. All other structures attached to the diaphragm have a tendency to move in a line parallel with the long axis of the body. A variable degree of lateral motion is permitted. The structure is uniform in consistency, outline, and surface, which constitutes a true hyperplasia in the clinical sense. Unless the spleen is dislocated it is impossible to grasp its upper margin, but the palpating finger rests on a smooth surface, which seems to be a part of a structure which extends upward beneath the costal arch. Its inner and lower margins are readily palpated, and the lower pole may often be grasped, which is characteristic of splenic enlargements. The inner margin is notched. The dulness on percussion over the structure in the abdominal region is continuous with that of a uniform dulness in the splenic region, extending often to the eighth rib in the post-axillary and the sixth rib in the anterior axillary line.

Tumors of the kidney are usually fixed, and their development from the lumbar region is evident on bimanual palpation. They develop, as a rule, downward, and, unless extremely large, seldom encroach upon the diaphragm. Urinary symptoms are often present, and the colon may be demonstrated as lying in front of the kidney mass.

Enlargement of the left lobe of the liver may present some difficulty in diagnosis. Under such circumstances the direction of the respiratory mobility, and, above all, the continuity of its lower border with the lower border of the liver in the epigastric region, would serve as distinguishing features. Having determined that the mass in the left hypochondriac region is a splenic structure, the nature of the enlargement must next be considered. It is characteristic of splenic hyperplasia that the form and outline of the normal spleen is retained. A neoplasm, such as carcinoma of the spleen, is to be excluded, or at least can scarcely be diagnosed in the absence of irregularities in the structure. Acquired syphilis rarely produces great enlargement of the spleen in the early stages of the disease, and still more rarely as a tertiary manifestation, excepting when combined with amyloid degeneration. The history, presence of amyloid elsewhere, and other signs of syphilis would render the diagnosis easy.

Enlargement of the spleen from tuberculosis would rarely present any difficulty in diagnosis. Echinococcus of the spleen is to be distinguished by its long course without general symptoms and the presence of fluctuation. In case of doubt, puncture would clear up the diagnosis. After having excluded all forms of neoplasm and cyst a hyperplasia of the spleen may be assumed. Its nature would then require careful consideration. A differential diagnosis should be made between the morbid

states which are accompanied by a splenic hyperplasia, namely, leukæmia, malarial cachexia, passive congestion, progressive pernicious anæmia, amyloid spleen, and Banti's splenomegalia with hepatic cirrhosis.

Splenic Leukæmia. The clinical phenomena are so nearly identical in the two affections that a blood examination shows the only essential difference between them. A slight increase in the polynuclear variety of leucocytes may be present in splenic pseudoleukæmia. In one of Banti's cases there was an absolute leucocytosis, but the blood examination was not made until the day upon which a pneumonia developed. In this and in a few other cases in which a moderate leucocytosis has been present the quality of leucocytosis has not been accurately stated. In the diagnosis of leukæmia, when leucocytosis is only moderate, the quality of the leucocytes, not their number, determines the diagnosis. Essential to the diagnosis of leukæmia with moderate leucocytosis is the increase in the mononuclear elements, which often show degenerative changes in their nuclei. As a rule, in splenic pseudoleukæmia there is a normal number of leucocytes, and the relation of the different forms is not disturbed. The number of red blood-corpuscles is at first moderately reduced. Later in the disease the number may be diminished to two million or one million, or, as in my case, to a little more than half a million per cubic millimetre. Change in size and form of the red blood-corpuscles is not marked. The individual red blood-corpuscles are poor in hæmoglobin and rarely contain nuclei. A more extended account of the blood in splenic pseudoleukæmia is to be found under symptomatology. The differential diagnosis between the clinical pictures presented by splenic pseudoleukæmia and splenic leukæmia is to be made only by a study of the white blood-corpuscles.

Malarial Cachexia. The form of malaria known as malarial cachexia, characterized by enlargement of the spleen and anæmia, presents clinical phenomena very closely simulating splenic pseudoleukæmia. Splenic hypertrophy, enlargement of the liver, and anæmic cachexia are essentially the same in both affections. Indeed, it must be admitted that a positive differential diagnosis between the two conditions could not be made under certain circumstances; as a rule, however, the differential features are sufficiently prominent to leave no doubt as to diagnosis. In all cases so far reported which are to be included in the category of splenic pseudoleukæmia, malaria has been carefully excluded as an etiological factor. A malarial cachexia, developing, as it sometimes does, in intense malarial districts, without chills, would present features requiring great care in diagnosis. In doubtful cases the presence of the plasmodium in the blood would, of course, be conclusive evidence as to the nature of the disease. Blood-pigment in the white blood-corpuscles, or free in the plasma, has not yet been noted in quantities approaching

that found in malarial cachexia. The pigmented skin is a slate or dirty yellow color, peculiar to chronic malaria. In splenic pseudoleukæmia pallor is combined with subicterus. In districts where malaria is intense a nephritis not infrequently complicates the disease, while it has never been noted in cases of splenic pseudoleukæmia. Inhabitants of a malarial district who have never had the usual manifestations of malaria, as well as those who unquestionably have suffered from the disease, may develop splenic hypertrophy, which often persists for a long time, even a period of years, without the appearance of anæmic cachexia or serious disturbance in general health. Splenic pseudoleukæmia is characterized by a splenic hypertrophy which is directly associated with or very soon followed by a progressive anæmia, which invariably continues to cachexia, with fatal termination in a definite period of time unless the disease is arrested. The therapy which in time will relieve a malarial cachexia has no particular effect on splenic pseudoleukæmia.

Essential Anæmia. In *chlorosis* the blood findings and clinical phenomena of the early course of the disease may be very similar to those of splenic pseudoleukæmia. However, in chlorosis the splenic enlargement is absent or seldom marked, and when present follows rather than precedes the development of the anæmia. The course and effect of treatment in the two affections is entirely different.

In *primary progressive pernicious anæmia* the cachexia, course, and clinical phenomena resemble the manifestations of this disease in many particulars, but the blood findings and splenic hypertrophy present differences sufficiently great to establish the diagnosis. In primary progressive pernicious anæmia the individual corpuscle is relatively rich in hæmoglobin, while in pseudoleukæmia it is poor. There is a much greater difference in the size and form of the red blood-corpuscles in primary progressive pernicious anæmia. Nucleated red blood-corpuscles are rarely, if ever, found in splenic pseudoleukæmia, while they are constantly found in primary progressive pernicious anæmia. The large form of nucleated red blood-corpuscle—the gigantoblast—of special importance in the diagnosis of primary progressive pernicious anæmia, has never been found in splenic pseudoleukæmia. In primary progressive pernicious anæmia the spleen is more often atrophied than enlarged. The enlargement, if sufficient to be detected on palpation, is never great, usually shows fluctuation in size under treatment, and always follows the development of anæmia. Mosler and Gast believe the enlargement of the spleen in chlorosis and primary progressive anæmia to be a compensatory hypertrophy directly connected with the regeneration of the blood. Other authors, as Immermann, Eichhorst, Müller, and Neumann, do not believe that under such circumstances the spleen returns to its prenatal hæmotopoitic func-

tion. By others the splenic enlargement in the anæmiæ is thought to be due to the increased destruction of red blood-corpuscles. The liver is nearly always hypertrophied in splenic pseudoleukæmia. In primary progressive pernicious anæmia it is not.

Passive Congestion of the Spleen. In this condition the etiological factors of a liver cirrhosis or other conditions inducing splenic hyperæmia would nearly always be evident. The course of the two diseases is somewhat different. The development of anastomoses between the portal and general circulation, and the other signs of liver cirrhosis, such as early appearance of ascites and late appearance of cachexia, characterize the spleen of passive congestion. The enlargement of the spleen in passive congestion is moderate in comparison to the enormous hypertrophy sometimes present in splenic pseudoleukæmia. In atrophic cirrhosis the liver is small, while in splenic pseudoleukæmia it is hypertrophied. On the other hand, cirrhosis of the liver accompanied by marked fatty infiltration and enlargement of the organ presents clinical features which under certain circumstances might give rise to great difficulty in diagnosis.

Amyloid Spleen. The diagnosis of amyloid spleen is to be made only when a sufficient cause has preceded its development, such as long-continued suppuration of bone or soft parts, phthisis pulmonum, or protracted syphilis. Evidence of amyloid degeneration elsewhere is to be desired in connection with its etiology. Although the spleen is usually the first organ to show amyloid changes, the diagnosis of amyloid spleen without signs of amyloid liver, kidneys, or intestines would be at best a probable diagnosis.

Splenomegalia with Cirrhosis of the Liver. Banti[1] describes a condition characterized by splenic hypertrophy associated with cirrhosis of the liver, in which he believes the primary disease is of the spleen, and that the cirrhosis of the liver results from the pathological changes in the spleen. The clinical difference between the affections is so slight that Banti admits that on that alone no distinction between the two affections can be made.

The diagnosis of splenic pseudoleukæmia is often surrounded by many difficulties. In all cases the evidence must be accurately obtained and the proper valuation placed on it to ensure trustworthy results. Undoubtedly, there are cases in which it would be advisable to withhold a positive diagnosis for a variable period of time.

Course and Prognosis. The disease presents a definite course within certain limits of variation. Its tendency is distinctly progressive. The severity of symptoms may show slight remissions; but, on the whole, it will be observed that the patient loses ground rather steadily from month to month. It is difficult to estimate the exact

[1] Semaine Med., 1894, xiv. 318, and Ziegler's Beitrage, 1898.

duration of the disease, owing to the uncertainty of the length of the first stage. In some cases a fatal termination has been reached in five or six months after the first symptoms were manifest. The average duration appears to be about two years. My case terminated in about three and a half years from the time the splenic enlargement was first noticed. That duration seems to be about the longest recorded. While the first and second stages of the disease may vary and be prolonged for a period of one or more years, the cachectic stage seldom continues more than a few weeks or five or six months. Death results from progressive marasmus and intercurrent disease. Medical treatment seems to be of no positive value in controlling the progress of the disease. The removal of the spleen has arrested the process in the cases noted in the chapter on treatment. The prognosis is decidedly unfavorable without surgical intervention.

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REVIEWS.

THE MEDICAL COMPLICATIONS, ACCIDENTS, AND SEQUELÆ OF TYPHOID OR ENTERIC FEVER. By HOBART AMORY HARE, M.D., B.Sc., Professor of Therapeutics in the Jefferson Medical College of Philadelphia, etc., with a Special Chapter on the Mental Disturbances following Typhoid Fever, by F. X. DERCUM, M.D., Clinical Professor of Diseases of the Nervous System in the Jefferson Medical College. Philadelphia and New York: Lea Brothers & Co., 1899.

CAN anything new be said about typhoid fever, a disease so widespread, so carefully observed, so critically studied, so voluminously written about, that knowledge concerning it would seem to be wellnigh complete and additional literature superfluous? Can even the industrious Hare glean from his own rich experience or that of others aught that is fresh concerning the medical complications of this preventable, disgraceful, filth disease? One must answer both no and yes. The facts concerning the atypical clinical manifestations of the affection, the numerous statistics, are largely gathered from authors who are dead or whose works are already well known. Louis, Fagge, Murchison, Liebermeister, Wunderlich, Immermann, Osler, and Fitz are men whose writings on typhoid are classic, though in some instances of recent date. There is, we might say, nothing new in this respect. But, on the other hand, there is much that is new unless one has kept in closest touch with the most recent literature. It is not so much, perhaps, the new fact that is of importance as it is the new point of view from which a fact is observed. Bacteriology has completely modified our conception of the disease. In its light even those dry old statistics have a glow of life. Instruments of precision and improved technique have made diagnosis surer. The Widal test, the Ehrlich reaction, the examination of the blood for leucocytosis or for bacilli clears up many a doubtful diagnosis. Pneumonia, cystitis, meningitis, cholecystitis, etc., long viewed as accidental or intercurrent affections, wear an entirely new aspect when viewed as localized manifestations of the working of the specific organism. Typho-malarial fever is banished or rightly looked upon as a double infection, typhoid and malaria; apyretic, mild, and abortive cases are more easily recognized, and cases that might escape the notice even of the morbid anatomist, because of absence of intestinal lesions, are made clear by the bacteriological test. It is, after all, the same old disease of Louis and Murchison and Liebermeister, with the same complications, but yet completely changed, because we have changed our point of view, even admitting that there have been some modifications in type as compared with the disease of the preceding generation, due to improved therapy and acquired relative immunity. To one who is not fully alive to the newer conception of typhoid, Hare's book is of the greatest value, for it treats of the subject in the light of the most recent knowledge.

As the reviewer reads the pages one after another he is forced to nod mental approval of both the statements of facts and conclusions reached, and to approve of the manner of expression. Though on the alert to find some faulty statement, he rarely has the fortune—good fortune from the critic's point of view—to be able to use the blue pencil of disapproval. Even grammatical and typographical errors are so scarce that it sent a chill—no, a typhoidal, cold, creepy sensation—up his back when he found one grammatical slip and detected a paragraph inserted several pages from its proper place (p. 163). This latter mistake leads us to speak of one of the merits of the work—viz., that it is quite up to date. Even as the book was going through the press several insertions were made, so that many references are to literature as late as April, 1899. Such a late "insert" has been put in (p. 163) under the head of thrombosis and embolism, though properly coming under the heading of hysteria.

There has evidently been a most careful study of standard works on medicine, and of monographs and special articles on typhoid fever, and a ransacking of indexes for facts and illustrative cases. There is liberal citation of authorities, free use of histories of cases of others and of one's own. In most instances references are given. So numerous are these and so exact that the book is really extremely valuable for its bibliography of typhoid. For while the author would not claim that a complete bibliography of typhoid complications and sequelæ is to be found here, yet it is no exaggeration to say that the chief references are here given, and one that would readily lead to others of a similar sort.

If one were disposed to be hypercritical one could pick out here and there abstracts of articles or histories of cases that might with advantage be condensed. We cite one instance: The unusually excellent article of Eshner on "Orchitis as a Complication of Typhoid" deserves mention, and his views should receive marked attention, but it would seem better to give his main points and conclusions, and to omit the table. Any one we believe interested enough to consult the table will look up the original article.

Again, an occasional doubt as to a correct diagnosis arises in the mind of the reader as to some of the odd atypical cases of typhoid where there is absence of autopsy, Widal test, rose-spots, or some fairly positive evidence.

It is impossible in treating a subject of this character to stick closely to the text, and our author, as he himself says, has found it difficult to avoid stepping over the dividing line between the ordinary symptoms and manifestations of the disease and those that are unusual or complicating. And we agree with him that a complete discussion of a complication may necessitate the consideration of the typical or common phenomenon of the malady. Once in a while, however, it has seemed to us that he has strayed a little further than was necessary or even wise—*e. g.*, on pages 79 and 80, where he discusses diagnosis. And to our mind the chapter on "Diseases that Ape Typhoid Fever" should be omitted entirely or enlarged. As it is, it is too sketchy and outlinear to be of more than suggestive value or to conform to the thoroughness and completeness of the rest of the book.

We have noted but few omissions, few statements of fact from which we would dissent. Diphtheria as a complication is not specifically mentioned (p. 110), though membranous pharyngitis is referred to. Cases

of diphtheria complicating typhoid are on record with bacteriological and post-mortem demonstration. We wish more emphasis had been laid upon the slow pulse as the rule in ordinary typhoid. Even though Liebermeister, Leube and many others have declared it to be of extreme value from a diagnostic and prognostic stand-point, many authors to-day lay, as it seems to us, too little stress upon the pulse that is slow in spite of high temperature, as the normal condition in the average typhoid. When one goes through a ward and sees on one temperature chart after another a pulse of less than 100 with temperatures between 102° and 105°, and this continuing even to convalescence, one realizes that Liebermeister is justified in saying that a good pulse in typhoid fever rarely rises above 110. This fact is mentioned by Hare, but not with the emphasis that it warrants, at least so it appears to us.

On pages 121 and 122 the question is discussed as to whether diarrhœa is indicative of a mild or severe infection. Hare in reality leaves the question open, saying: "In all probability diarrhœa is neither indicative of a severe nor a light attack in many cases, although if it be violent the exhaustion produced by the discharges may seriously imperil the patient's chances of recovery." The writer has learned to dread the cases with diarrhœa; cases with constipation cause far less anxiety. And we dissent from the statement that tympanites is less frequently present in cases with active diarrhœa.

The work is systematically arranged. After a preliminary chapter on "General Considerations" there are discussed varieties of onset, the aberrant symptoms, states, or complications of the well-developed stage of the disease, the complications of the period of convalescence, the conditions which ape typhoid fever, duration and immunity to second attacks, the mental complications. In each chapter there is an orderly treatment of the complications as they affect the various organs or systems of organs of the body.

The last chapter, on "The Mental Complications," is written by F. X. Dercum, and is a clear portrayal of the mental conditions associated with typhoid fever, conditions too often loosely grasped by the practitioner under the comprehensive term of typhoid or post-typhoid insanity. The dividing line between the different varieties is clearly drawn by Dercum, and he emphasizes what has already been previously referred to by Hare in other places, that overmedication may at times produce complications or conditions that simulate them. Extremely interesting are the cases of Dercum's (pp. 225 and 271), where the too free use of alcohol induced typical alcoholic neuritis, with, in one case, delirium. Overmedication and overfeeding may likewise be held accountable for some of the gastro-intestinal disturbances that are sometimes wrongly attributed to the disease.

No one can fail to be interested in reading this book, and even though well up in the current literature of typhoid one will profit by the reading. It is a good thing to compare notes, and there is no physician of even a few years' practice who cannot, as he reads, make mental comments by comparison with his own experience. Judged by this test, always a severe one, as well as by other tests, the work deserves the highest praise. It treats the topic from a modern, scientific stand-point, is rich in illustrative cases, contains ample literary references, and is full of suggestive practical hints. J. B. H.

A Text-book of Pharmacology and Therapeutics on the Action of Drugs in Health and Disease. By Arthur R. Cushny, M.A., M.D. Aberd.; Professor of Materia Medica and Therapeutics in the University of Michigan, formerly Thompson Fellow in the University of Aberdeen, and assistant in the Pharmacological Institute of the University of Strassburg. Illustrated with 47 engravings. 8vo. pp. 730. Philadelphia and New York: Lea Brothers & Co., 1899.

This volume of Dr. Cushny's on Therapeutics is dedicated to Oswald Schmiedeberg, of Strassburg. The introduction deals with the mode of action of drugs, their elective affinity, their remote and local effects, their chemical composition and pharmacological action, the conditions modifying the effect of drugs, methods of administration, clinical characters of drugs, and their classification. In this part the author states that the scientific clinician is imbued with the desire to ascertain the method in which drugs act as well as to cure disease, and thus unite clinical observation with pharmacological research. He believes that laboratory experience and the bedside results are necessary to a complete knowledge of the action of a drug. In his classification of drugs he follows Bucheim and Schmiedeberg, and divides them into six kinds. In the first division we find organic substances, which are characterized chiefly by their local action. This includes the demulcents, emollients, sugars, and flavoring substances, simple bitters, volatile oils, skin irritants and counter-irritation, vegetable purgatives, vegetable astringents, and anthelmintics. The second division of drugs is organic substances characterized chiefly by their action after absorption. In this part he takes up alcohol, holding with his teacher, Schmiedeberg, that it does not excite but depresses the central nervous system from the beginning. He regards alcohol as a food, for in undergoing combustion alcohol gives energy to the body, and therefore is technically a food. He states that alcohol acts as a substitute for fats and carbohydrates in the food to some extent. As to the action of chloroform, the author believes that all exact experiments of the last few years show that the respiration fails before the heart, and that from a practical point of view it is of comparatively little importance whether there are a few fluttering beats of the heart after the last inspiration or not. The all-important question, he believes, is whether the heart has been so injured as to be unable to carry on the circulation, and this is decided by the concentration of the vapor that has been inhaled. For strychnine-poisoning he advises the inhalation of chloroform instead of the administration of chloral. The advantage of the anæsthetic over the chloral is that it can be stopped if any symptoms of strychnine paralysis appear. In opium-poisoning he advises the use of caffeine, either hypodermatically or in the form of strong coffee by the stomach. He also favors the use of atropine, but only in small doses, as large ones depress the centre of respiration. He thinks caffeine preferable to atropine, as it is scarcely possible to paralyze the respiratory centre with it. In treating of ipecacuanha, he writes that it was formerly largely used as an emetic, but is less suitable for this purpose than apomorphine, which acts much more rapidly, especially when injected hypodermatically, and which is followed by less nausea and discomfort. I fear the majority of the profession will employ ipecacuanha in preference. In the third division of drugs, he takes up the combination of the alkalies, alkaline earths,

acids, and allied bodies. In treating of calcium salts the author states that rickets is not due to a lack of lime in the food, nor, in fact, in the tissues generally, but to some abnormal condition which prevents the lime salts from being deposited in the bones, although they are present in abundance in the blood. The author holds that from this fact it follows that lime salts are not likely to be of benefit in rickets (and the same holds true in osteomalacia), unless when it is due to lime starvation, a condition which is unlikely to arise in the human subject. The author does not think that the lime treatment of hæmophilia and aneurism is of much value, for it would seem to be very improbable that the absence of coagulation is due to deficiency in the lime salts, for much more is taken in the food than is sufficient for the organism, and the pharmacopœial salts are not more easily absorbed than the combinations present in food. In the fourth division he takes up the heavy metals. He holds that when a solution of a metallic salt comes in contact with a living tissue, such as the mucous membrane of the mouth or stomach, the albuminate is at once formed, and the acid with which the metal was combined is set free. The character of the local action of the salt depends upon these two factors—the nature of the precipitate and of the acid. The author finds that the most powerful corrosive salts of any metal are those in which it is combined with the corrosive rapidly dissociated acids—that is, the chlorides and nitrates, provided they are soluble. The least corrosive of the salts of the metals are those formed with the slowly dissociated organic acids, such as the acetates. In the fifth division of drugs he has the ferments, secretions, and toxalbumins. In this section he gives the latest views upon the internal secretions, speaking of iodothyrin, of the thyroid and of epinephrine of the adrenals. In the sixth division he takes up the mechanical remedies.

In this work the physiological action of a drug is not treated so much *in extenso* as in Dr. H. C. Wood's *Therapeutics*—only the results of investigations are given. In the literature of physiological experiments, he seems to have chiefly consulted German authorities, for many original American contributions have apparently been overlooked. The part of the work on the application of remedies to disease is quite full and up to date; while there may be some dispute about the author's estimate of certain drugs in disease, yet he always gives reasons for his views.

Dr. Cushny has ably performed his task of collecting and bringing together the opinions and experiences of the most eminent writers as to the real value of the articles of the materia medica in the treatment of the disease. Taking the work as a whole, it displays considerable originality and does great credit to the author. He seems to have included all the drugs used at the bedside, even heroin. It is a very suitable text-book for students, and will be of considerable advantage to the busy practitioner, to keep abreast of the very latest advances either in the laboratory or at the clinic. I. O.

THE MECHANICS OF SURGERY, comprising Detailed Descriptions, Illustrations, and Lists of the Instruments, Appliances, and Furniture Necessary in Modern Surgical Art. By CHARLES TRUAX. Chicago, 1899.

THIS is a large volume of over a thousand pages, and at first sight appears like an instrument-maker's catalogue, minus the prices. The

author states that his aim has been "to illustrate and describe such mechanical appliances as research and experience have proved to be suitable or best adapted to the purposes for which they were designed." This he has done in a fairly acceptable manner. Also, he states that text-book descriptions are usually unsatisfactory, but that "the instrument-maker, on the other hand, who happens to be of a mechanical turn of mind and fairly well informed in operative surgery, can usually give judicious advice in the selection of instrument patterns." This is more true of the inexperienced than of the experienced surgeon. Another of the author's objects is to secure a standard nomenclature for surgical instruments. For this effort he is deserving of all praise. He thinks there is a place in the library of every practitioner of medicine or surgery for such a book, and states that "if this volume fails to fulfil the expectations of its author, it may perhaps serve as a stimulus to some abler mind to prepare a work which will better meet all requirements." The author evidently appreciates the fact that there is wanting a work which will elucidate the mechanics of surgery. That this is so there can be no doubt. That the book has taken the form it has is consequent on the fact that its author is an instrument-maker and dealer. Instead of a treatise on principles of mechanics as applied to surgery, we have detailed descriptions of instruments; instead of discussing the best means of accomplishing an object, he gives a description of the various appliances which are used. What would be of service is his candid opinion of the relative merits of the various instruments, with his reasons therefor; but that evidently is too much to expect of one who is financially interested in the various instruments he describes. The author evidently appreciates what should be done, for we get glimpses of his possibilities in his discussion of the manner of holding a needle and in his short comments on the mode of use of various instruments. He knows, but he has not the courage to state his convictions. Personally, we do not think that the book is scientific enough to deserve success; it still breathes too much of the trade spirit. Surgeons want a book written from the stand-point of a user not a seller of instruments; they want to be informed as to utility not fashion. Principles live, but fashions change. The book is, however, much better than a mere catalogue, and the author deserves some praise for taking a step in the right direction, even though it be not so decided a one as the subject demands and the author himself is capable of making.

G. G. D.

La Vie sur les Hauts Plateaux (Life on High Plateaux). Par Le Prof. A. L. Herrera et Le Dr. D. Vergara Lope, Anciens Presidents de la Societe "Alzate," Mexico. Awarded the Hodgkin's Prize silver medal of the Smithsonian Institution, Washington, D. C., 1895. Royal octavo, pp. 792. French translation with numerous plates. Mexico, 1899.

This immense work is dedicated to President Diaz, of Mexico. It will be remembered that the first prize of $10,000 was awarded to Lord Rayleigh and Prof. William Ramsay, of London, for their discovery of argon in the atmosphere. The Mexican work seeks to demonstrate what seems to be a fact, that both vegetable life and animal life attain a higher degree of perfection at the high elevations found in the plateaux

of Mexico. In both men and the lower animals tuberculosis is very rare. In 1885, out of 70,000 cattle killed in the general abattoir of the city of Mexico, after very careful examination, 45 only were found to be tuberculous. The authors give a good description of the country, the distribution of plant-life, with especial reference to altitude, the influence of climate on the development of plants, the vertical distribution of vertebrates and the effect of high altitudes upon them. This portion of the work is copiously illiustrated with engravings of various domestic animals, and makes a very interesting portion of the volume. The authors then take up anthropometry and the physiology of man in the higher altitudes, with statistics and other observations on the prevalence of various diseases. The higher elevations are proved beyond question to be far more healthy than other portions of Mexico. The mortality caused by tuberculosis is four times greater between sea-level and 500 metres than between 500 metres and higher levels. In the City of Mexico, elevation 7000 feet (2268 metres), the deaths from tubercular disease in 1892 were about 8 per cent. of the total mortality.

The results of this investigation show that the composition and general character of the blood are altered by residence in high altitudes. The corpuscles increase in number, the urine becomes more dense, less abundant, and has a greater proportion of urea. The number of respirations and pulsations of the heart increase; so do the respiratory capacity and the mobility of the chest-walls. There is also a diminution of the intravascular blood-tension. They account for the augmentation of the number of red blood-corpuscles per cubic millimetre by attributing it to the increased density of the blood due to the extreme dryness of the air. This raises the count to between 6000 and 7000 per cubic millimetre, and thus they corroborate the findings of Solly, who has pursued similar investigations at Colorado Springs, elevation 6000 feet. Studies of this kind are highly appreciated at the present time, when commercial and political interests throughout North America are more closely bound together. G. H.

CHEMISTRY: GENERAL, MEDICAL, AND PHARMACEUTICAL, INCLUDING THE CHEMISTRY OF THE U. S. PHARMACOPŒIA; A MANUAL ON THE SCIENCE OF CHEMISTRY AND ITS APPLICATIONS IN MEDICINE AND PHARMACY. By JOHN ATTFIELD, F.R.S. Sixteenth edition. Philadelphia and New York: Lea Brothers & Co., 1899.

THE successive editions of this excellent work have been reviewed in these columns, and the present edition merits the continued praise of the reviewer and laudation of the reading public. Written from the chemical stand-point, this treatise offers a fulness of detailed information upon the chemical aspects of medicine and the collateral sciences, and gives, in fact, as much as any book which is written from the stand-point of the chemist, and not of the physiologist and pathologist. As in all books of this character, the inorganic predominates over the organic chemistry; the sections upon the latter are, however, exceedingly well written. Such a treatise is indispensable to the alert practitioner as a work of reference, to the student as a guide, and to the scholar as a companion. A. E. T.

THE RETROSPECT OF PRACTICAL MEDICINE AND SURGERY. Being a Half-yearly Journal, Containing a Retrospective View of Every Discovery and Practical Improvement in the Medical Sciences. By JAMES BRAITHWAITE, M.D. Lond., assisted by E. F. TREVELYAN, M.D. Lond., B.Sc., M.R.C.P. Vol. CXVIII. January, 1899. Uniform American edition. New York: G. P. Putnam's Sons, 1899.

MEDICAL literature has its parasites, and these are the feeders of parasites of the second degree. The present volume, like its predecessor, belongs to both parasitic groups. How valuable the collection of reprints and abstracts from various sources—good, bad, and indifferent—is likely to prove to the general practitioner we are unable to surmise. Unquestionably, if a man reads no other medical literature he will obtain a great deal of valuable information from Braithwaite's retrospect; but if he desires to keep abreast of medical progress, and to know what is best in the medical thought of the day, we fear that he will be greatly disappointed by the material furnished by the editors. The first half of the book is called the synopsis. It is difficult to understand why it exists at all, for the material could be much more satisfactorily distributed in the second or systematic portion. It consists entirely of abstracts taken from various sources, and usually representing papers that are not of the highest value. There is a certain degree of classification into medicine, surgery, etc., that is well as far as it goes, but under each subheading the articles are arranged alphabetically, which produces a rather incongruous juxtaposition at times. The second part of the book is very much better than the first. Extracts, not abstracts, are given from 130 articles representing the whole domain of medical science. We cannot altogether approve of the selections. Many articles of considerable value have been omitted, and many others of considerably less importance inserted. The editors appear to have been attracted rather by theoretical or statistical material than by actual clinical or pathological facts and observations. German and French literature, to say nothing of that of other countries, has been almost entirely neglected, the few articles included being, as is frequent in the English and American journals, not always the most important. The publishers have done their part creditably. The book is well printed and convenient to hold. The proof-reading, as a whole, is excellent. A few absurd mistakes have, however, been passed (page 33). It would seem reasonable to suppose that in the multiplicity of books of this type the most desirable arrangement would have been discovered, and would survive by a process of natural selection. There are, however, almost as many methods as books, and none seem to be satisfactory. The present book is probably adapted to the wants of a certain number, and although neither perfect nor even the best, is not wholly without good points. J. S.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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Tabes Dorsalis.—THOMAS (*Bulletin of the Johns Hopkins Hospital*, April, 1899, p. 51) has analyzed 111 cases of tabes which have been treated in the Johns Hopkins Hospital and Dispensary since its opening in 1889. Of the 111 cases, 106 were white and five negroes. Thus only 4.5 per cent. of the cases were in negroes, although the latter constitute 10.05 per cent. of the new cases treated. This low percentage is remarkable when one considers that of the cases of early syphilis treated in the genito-urinary dispensary during the past two years the number was five and a half times greater in the negro than it was in the white race. This would seem to diminish the importance of syphilis as an etiological factor.

Males were affected much more frequently than females. Ninety-seven cases were in males and fourteen in females. The females were all white.

Most of the cases developed between thirty and fifty. The youngest case was twenty-five and the oldest sixty-six. Fifteen cases developed after fifty years of age.

The history of syphilis was carefully studied. The results were: Certain syphilis, 42.1 per cent.; possible or probable syphilis, 63.1 per cent. "These figures fall below those obtained by many of the later observers, but it is not due to lack of zeal."

Regarding the bearing of syphilis as an etiological factor in this series, Thomas says:

1. In a large proportion of cases of tabes a history of syphilis can be obtained.

2. In a certain and a not inconsiderable number of cases there is no history of a venereal sore or other syphilitic manifestations.

3. In negroes tabes is relatively uncommon, whereas syphilis is much more common in them than in the white population.

4. The partial immunity of women is greater than can be satisfactorily accounted for by the relative infrequency of syphilis among them.

Thomas believes that the study of his series would seem to show that syphilis is an important factor in the etiology of tabes, but that it is far from being so important a factor as many would wish to make. That syphilis is not the only factor seems to be shown.

The Iodine Reaction in Leucocytes.—KAMINER (*Deutsche med. Wochenschrift*, 1899, No. 15) has made a critical study of the iodine reaction in the leucocytes, and opposes the idea usually accepted that the brown color depends on glycogen, as Ehrlich and others claim. Kaminer describes three degrees or stages of the reaction. The first is that of diffuse staining; the protoplasm is reddish, the nucleus unstained. In the second, more or less intensely brown granules and masses can be seen. In the third, rarely seen, the protoplasm is wholly converted into dark brown masses, the nucleus still unstained. Free masses showing the reaction were rarely seen, but the author does not agree with Czerny that they are artefarcts. In normal individuals the author never observed the reaction. In disease there was no definite relation between the occurrence of the reaction and the pathological process. In puerperal fever, with or without suppuration, it was always present, but in other diseases, febrile and inflammatory, it was sometimes present and sometimes absent. When present the reaction varied in intensity without regard to the other clinical features. It was never present without leucocytosis. In sepsis all stages were present. In pneumonia it was usually present, but never in the third degree. In phthisis it was present at times, but only in the first degree, and in rheumatism it was never obtained. Leucocytosis was sometimes observed without the reaction, and the latter was present in cases without localized suppuration. Of special interest are the results of the test in diabetes. In four cases there was never a trace of reaction. In these there were no leucocytosis. In the fifth case, examined in coma, with acetonuria and leucocytosis, the reaction was present in the second degree, with extracellular granules. Unlike Czerny, the author never obtained the reaction in anæmic and cachectic patients, but in many cases of cancer with necrosis or other complications he had positive results. In a case of hemorrhagic diathesis with leucocytosis the reaction was obtained. Agents that increase the leucocytes in the blood, such as spermin, did not affect the reaction, nor did aseptic fever cause it. Toxic substances, on the other hand, in rabbits, seemed to increase the iodine reaction of the leucocytes. The author concludes that the substance giving the reaction is a product of degeneration formed in the leucocytes and not taken up from other organs.

Recklinghausen's Disease, with Sarcomatous Growth Involving the Brachial Plexus.—ROLLESTON enumerates the cardinal signs of the morbid entity, described first by VON RECKLINGHAUSEN as being: 1. Tumors of the skin of the nature of molluscum fibrosum. 2. Subcutaneous tumors situated on the nerves plexiform or fibroneuromata; these are multiple and usually small-sized, and may be on peripheral nerves, on larger nerve-trunks, near the nerve-roots, on the cranial and sympathetic nerves. 3. Pigmentation of

the skin, either patches of considerable size or small specks resembling freckles. In addition there may occur pain, arthralgia, alteration of sensation, and impaired mental activity. The term generalized neurofibromatosis has been applied to the disease.

Rolleston reports a case of a man, aged thirty-two years, having a large patch of molluscum fibrosum on the left shoulder from birth. The skin of the body was generally brownish, but there were irregular areas of pigmentation, some as large as a shilling, the majority, however, resembling freckles. Under the skin of the left back were a number of small, firm, readily movable tumors appearing to be multiple neurofibromata; there were a few similar nodules under the skin of the right side of the abdomen; no diarrhœa was present.

The further interest in the case was the development of a large tumor on the left side of the vertebral column at the base of the first dorsal vertebra, giving rise to such pressure symptoms as contraction of the left pupil, ptosis, increased pulse-rate, ulnar nerve paralysis, and, later, dyspnœa, stridor, and brassy cough. An exploratory operation revealed too extensive a tumor to allow removal; pieces removed for examination showed it to be spindle-celled sarcoma.

At autopsy a very extensive growth could be made out involving the inner cords of the brachial plexus; the tumor had spread into the thorax and had occupied the superior mediastinum on the left side. Surrounding structures, the internal jugular vein, trachea, left bronchus, and thoracic duct were compressed, and, in the case of the vein, obliterated; the cervical sympathetic was extensively destroyed.

Rolleston suggests that the tumor had its origin in a neurofibroma, becoming subsequently a malignant neuroma.

The nodules under the skin of the back and abdomen were fibroneuromata, with some myxomatous tissue; neuromata were present in the abdominal sympathetic.

Various theories have been put forth in explanation of von Recklinghausen's disease. FEINDEL suggests that it is the outcome of a congenital malformation of the ectoderm, and is supported by OPPENHEIM. To Rolleston it seems more probable that a developmental rise on the part of the mesoblast in the corium and nerves is responsible, this accounting for the fibroneuromata and the molluscous growth, while the pigmentation of the skin is allied to the common pigmented moles that occasionally develop in malignant melanotic growths. Rolleston mentions, further, the fact that GOLDMAN and BOWLBY describe sarcomatous changes in neuromata.—*Lancet*, July 29, 1899.

The Excretion of Pepsin in Achylia Gastrica; the Estimation of Pepsin in the Urine.—TROLLER (*Archiv f. Verdauungs-Krankheiten*, Band v. Heft 2) records two cases that would usually be classed under the head of achylia gastrica, since free hydrochloric acid was entirely absent in both cases for a considerable period. One patient in especial had had digestive disturbance for nine years, had very low total acidity and complete lack of free hydrochloric acid, while the gastric contents showed after rough testing no power of digesting albumin. Troller made more accurate investigations concerning

the presence of pepsin, however, and found that by a modification of Hammerschlag's method he could determine that the gastric contents did digest albumin in both cases. From these results he says, with somewhat rash decision, that achylia gastrica is a misnomer for most of these cases, and that the term hypochylia should be used instead, since, though hydrochloric acid is absent, ferments will be found if the examination for them is sufficiently careful. His report concerning the presence of pepsin in the urine is of greater interest. He modified Hammerschlag's method for the quantitative determination of pepsin by using protargon instead of fibrin in making up the solution used as a test of the digestive power, choosing protargon because it dissolves much more completely in a hydrochloric acid solution, and greater accuracy of results is, therefore, possible. He then investigated normal urine, and found in all instances small quantities of a substance that digested albumin in an acid medium, and which he, therefore, believed to be pepsin. Then cases of gastric disease were investigated, and it was found that the quantity of pepsin in the urine varied directly with the condition of the stomach contents. Hence, he decides that if the use of the stomach-tube is contraindicated the quantitative determination of the pepsin in the urine will give a direct indication of the secretory power of the gastric glands. The proteolytic action of bacteria is believed to have been entirely excluded by the fact that all of the solutions of protargon used as tests contained free hydrochloric acid. (The number of cases reported is small, however, the method is subject to serious error, and the results are at present merely interesting, not conclusive.)

The Excretion of Uric Acid during an Acute Attack of Gout.—Badt (*Zeitsch. f. klin. Med.*, Band xxxvii. Heft 5 and 6) delivers another blow at the old and generally accepted teaching concerning the relation between uric acid excretion and gout. He reports the results of estimations of uric acid in five cases of gout, four estimations being made in each case, three of them during the acute attack, and the fourth some weeks later, in order to compare the conditions at the two periods. In no instance was the excretion found lessened during the attack; indeed, it was usually greater than in the interval, though the increase was so slight as to be negligible. This is, of course, directly opposed to the old teaching that there is uric acid retention in the early part of the attack, and free excretion as the paroxysm passes off; and the work is confirmatory of that done by other recent workers who have used accurate methods. Taylor's work (The American Journal of the Medical Sciences, August, 1899) has shown very convincingly that in a healthy man the excretion of all the purin bodies is almost exactly parallel to the quantity contained in the food, a fact which has been generally overlooked in considering the amount excreted in attacks of gout. This is a matter of especial importance, as but small quantities of food are taken, as a rule, in the early part of the attacks, and as the intensity of the suffering and depression becomes less the patients take relatively larger amounts of food.

The Influence of Nutrient Enemata on the Gastric Secretions.—Ziarko (*Wiener klin. Wochenschrift*, 1899, No. 18) publishes some investigations on this subject. The statements of previous observers have been contradic-

tory; exact examinations were lacking, and some have gone so far as to assert even that rectal feeding is not useful in the treatment of gastric ulcer, because it stimulates the flow of hydrochloric acid. The author examined the stomach contents before and after giving nutrient enemata in ten persons with various diseases. The result was that within two and one-half hours after the injection there was no increase in the amount or acidity of the gastric contents. In fact, the acidity was reduced, due, the author thinks, to the secretion of mucous.

A New Focus of Ankylostoma and Its Peculiarity.—ZINN and JACOBY in their work upon the geographical distribution of ankylostoma duodenale omit a focus of the disease located on the island of Madeira. The attention of Dr. J. Goldschmidt was first directed to this new focus by a landowner of Porta da Cruz, Northern Madeira. His tenants and workmen were attacked by a peculiar disease known among the people as "ictericia," *i. e.*, jaundice, which had been introduced by a returned islander who had been absent in Brazil for many years. During the first five years there were five fatal cases. The diagnosis of ankylostomiasis was made by Dr. Vincent Machado, and confirmed by Prof. Leichtenstern. The cases were all among the poorest class of peasants and among those crowded into dirty huts; entire families were often infected. Without exception people living under favorable circumstances remained free from the disease. Fatal cases, which were not so rare at first, have not occured during the past four years. Across the mountains to the south, in the village of San Martinho, there was another focus, and these seem to be the only ones on the island. Tænia, oxyuria, and ascaris are very common in all parts of Madeira.

The author draws the following conclusions: Ankylostoma is carried through great distances by infected persons. The possibility of infection depends upon the close contact of healthy with infected men. For the existence of an ankylostoma epidemic it is necessary that many poorly nourished men ingest these worms without having their growth in the bowel disturbed by too many other parasites. In weak individuals a few worms will produce the symptoms of ankylostomiasis, while in the more robust it requires a large number. By administering anthelmintics, such as thymol and felix mass, an epidemic can be stopped.—*Deutsche med. Wochenschrift*, No. 14, 1899.

The Blood in Hysteria and Neuroses.—LUXENBURG (*Centralblatt f. Inn. Med.*, 1899, No. 21) reports a series of examinations in forty cases. The examinations included counts of the corpuscles and estimations of hæmoglobin, fibrin, water, and total solids. The results showed: 1. That in contradiction to the usual view, and notwithstanding the anæmic appearance so frequent in patients with functional neuroses, there was in these cases no hydræmia. 2. The red blood-corpuscles were often considerably increased, considered by the author to be the result of vasomotor disturbance at the time of the examination. 3. The leucocytes were often subnormal. 4. The fibrin often showed marked changes.

Acute Graves' Disease.—HIRSCHLAFF (*Zeitschrift f. klin. Med.*, 1899, Band xxxvi. p. 200) reports a case of great interest. The patient was a woman,

aged twenty-one years, who for nine years had had attacks of palpitation of the heart and dyspnœa at the time of menstruation. In 1895, after a fright, she had convulsions and became unconscious, and similar attacks occurred every month, preceded by paræsthesia of the hands and feet. Excitement and anger frequently brought on the attacks. Three weeks before admission to the hospital the patient's skin became dark, she grew weak, had constant headache, and complained especially of palpitation of the heart. The eyes became prominent. The appetite was increased, but there was steady and marked loss of weight. In addition to the symptoms mentioned, the patient complained of weakness, with giving way of the legs, and excessive night-sweats. On admission there was general pigmentation of the skin, most marked on the extensor surfaces, the axillæ and groins, areolæ, and external genitals. The perspiration was excessive. Exophthalmos was marked, with Graafe's, Stellwag's, and Mœbius' symptoms. There was great restlessness, almost amounting to choreic movements, and a fine tremor of the hands. The patient was very excitable. The thyroid was slightly enlarged. The heart was enlarged, the sounds pure; pulse 100. The urine and blood were negative. While under observation the patient gained in weight, the pigmentation became paler, menstruation reappeared. A number of hysterical attacks occurred, following trifling annoyances. The temperature grew irregular, the stools became diarrhœal, and after a sudden and unexplained rise of temperature, with frequent pulse and respiration, the patient died. Autopsy showed adenoma of the thyroid, hyperplasia of the thymus, tonsils, spleen, and of the adenoid tissue in the stomach and intestines, and hypertrophy and dilatation of the heart. As infection could be excluded (?), the author looks on the changes in the lymphatic tissue as due to an intoxication, the same that caused the fever and the sudden death.

During life the metabolic processes were carefully studied, the period of observation—forty-six days—being the longest so far reported in this disease. The results are given in detail. The medication, including thymus and thyroid tablets, had no effect on the disease or the metabolism, but was used for so short a time that no definite conclusions can be drawn, especially in regard to the harmlessness of the thyroid extract. The emaciation was overcome by overfeeding, but aside from a few symptoms this had no effect on the disease as a whole.

Pneumonia with Peculiar Course.—Pychlau (*St. Petersburger med. Wochenschrift*, 1898, No. 52) reports the following case. Fourteen days after a severe attack of angina, the patient, a child, developed a temperature of 39.5° C., pulse of 160; the breathing was weak and rapid; there were vomiting and marked apathy. On examination of the chest distinct bronchial breathing was detected in the right lower lobe. During the night there was profuse sweating, followed by profound sleep and a fall of temperature to 36.6° in the morning. The breathing was quiet, the pulse regular, 84; there was neither cough nor bronchial breathing, but at midnight the temperature rose to 41.1°; the pulse was 130 to 140 and the breathing 50 per minute. Over the section of the lung first affected the murmur was purely vesicular, while in the right upper lobe there was indistinct bronchial breathing, with a somewhat tympanitic note on percussion. On the following morning the

temperature was 36.6°; there was complete euphoria, with no signs in the lungs. In the evening the temperature again went up, reaching 41°. From this time there was distinct infiltration of the left lower lobe, with characteristic signs. Ten days after the infiltration of the left lower lobe was first demonstrated the temperature was for the first time under 37° C. It rose the next day, however. On the nineteenth day of the disease and the seventh day of the second period of fever crisis occurred, the temperature falling to 36° C. The author believes that this case was a mixed infection, a part of the symptoms being caused by Fraenkel's diplococcus and others by the streptococcus. He is in doubt as to whether this was a case of croupous pneumonia, with delayed and somewhat unusual course and very tardy crisis, or a recurring pneumonia in which the first crisis occurred at the beginning of the twelfth day of the disease, and after only twenty-four hours' intermission the recurrence began. He also considers the possibility of a wandering pneumonia. The author thinks the pneumonia was secondary to the angina, the infective agents being streptococci and diplococci harbored in the tonsils. The child had not been out of the house for weeks, and there had been no pneumonia in the house for a year at least.

SURGERY.

UNDER THE CHARGE OF

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The Technique of Craniotomy; A New Conductor for the Wire Saw.—PODREZ (*Centralblatt für Chirurgie*, 1899, No. 9), realizing the advantages of craniotomy when performed by the osteocutaneous flap method, with the use of the wire saw, has devised an instrument for passing it rapidly from one opening in the skull to another, and afterward protecting the brain and its membranes from injury while the bone is being divided.

The instrument is made from a watch-spring, 3 to 4 millimetres broad and 13 to 15 millimetres long. At one end is a ring or handle, at the other a rounded tip perforated with an eye, to receive the silk ligature that is connected to the saw, and is double the length of the conductor. The ligature is attached to the saw, then threaded to the middle in the eye of the conductor. Two openings are made at the ends of the line of incision, in a slanting direction, in which the saw is to be passed. The director is then passed, armed with its thread, and is made to emerge at the second opening. The thread is removed from the eye and the saw drawn through. The guide, on account of the

recoil of the spring, presses the membranes of the brain and the brain itself inward, and prevents injury by the saw. Three sides of the square to be removed are cut in this manner. In the fourth only the inner table is cut, while the outer is broken, preserving the periosteum and skin pedicle.

On Nephralgia.—HUBBARD (*Annals of Surgery*, August, 1899) reports five cases in which no assignable cause could be found for the pain and with symptoms that were so severe as to simulate calculous disease. While holding that nephralgia is a term expressive of a symptom rather than a disease, he believes that there are a certain number of cases which must be classed under this term where pain is the most marked symptom.

All of the cases reported were operated upon, and in all but one a nephrotomy was done and the kidney searched for stone, without any being found. Four cases recovered from the pain and other symptoms, as hæmaturia and pyuria. The one that was not relieved had had the kidney incised, but no stone was found.

The symptoms are so often those of stone in the kidney that the differential diagnosis is difficult and rarely made. Age and sex are no help. Stone is found in the right or left kidney in relatively the same proportion of cases. Involvement of the testicle by the pain is a little more frequent in nephralgia. The urine gives the greatest help; it is free from blood, pus, and crystals in a great majority of cases, though it may be free in calculous disease. The urine may also be normal in those cases where the flow is stopped, at least temporarily, from the diseased kidney by the impaction of a stone in the ureter. However, in most cases where the symptoms are sufficiently persistent and severe to warrant the diagnosis of stone the urine will be found to be abnormal. With regard to distinguishing the urine of pyelitis from that of cystitis, it is said that the reaction in the former is usually acid while in the latter alkaline. Cystoscopy may be of aid in determining which kidney is diseased. The diagnosis from tuberculous kidney may at times be difficult.

Operation is usually started with the idea that a stone is present. To be thorough, the examination for calculus must be made with a probe as well as by the finger. If nephrotomy is not effectual, palpation and needling are of uncertain value. The ureter should be probed for calculi. If nothing abnormal can be found the capsule should be split from pole to pole and the kidney fixed in place by sutures before closing the wound. The prognosis, though not always favorable as to cure, is certainly, in the light of present experience, much better than formerly, and if any mobility has been detected and rectified relief may be hoped for.

The Treatment of Injuries of the Spinal Cord.—BOLTON (*Annals of Surgery*, August, 1899) makes the following summary of his method of treatment:

Extradural hemorrhage does not give rise to cord lesions or symptoms, and requires no treatment.

2. Total lesions of the cord are irremediable, because the cells and fibres of the entire cord are destroyed, are never regenerated, and are replaced by cicatricial tissue. The lesion thus is permanent, and requires no treatment.

3. In hæmatomyelia the clot is absorbed; its site persists as a cavity or is filled by newly-formed tissue; irregularities of circulation in the surrounding portions of the cord adjust themselves. There may be great amelioration of the symptoms. There is, therefore, no therapeutic indication, and no remedial treatment is possible.

4. In partial contusion of the cord the lesion results in permanent destruction of cells and fibres; disturbances of circulation adjust themselves. Repair is accomplished by cicatricial tissue. No treatment is available.

5. In open injuries of the cord there are destruction of cells and fibres and disturbances of circulation. In addition, infection may occur or a foreign body be introduced and left in or lodged against the cord, and by its continued presence produces great disturbance of circulation and consequent extensive degeneration and necrosis of cells and fibres. Repair occurs by cicatricial tissue. Here immediate operation is indicated for antisepsis, drainage, and the removal of the foreign body.

Observations on the Sterilization of Catgut —DARLING (*Journal of the Boston Society of Medical Sciences*, May, 1899) has shown by a series of experiments that it is possible and practicable to sterilize catgut by dry heat and yet maintain the tensile strength of the gut. When baked in the ordinary way it becomes shrivelled and brittle if the temperature is high enough to sterilize it. This may be effectually prevented by wrapping the separate strands in several layers of paraffin paper. This simple procedure prevents the driving out of the moisture and fat in the catgut, so that it remains unshrivelled and flexible after a prolonged exposure to dry heat. This use of paraffin paper is not new; it was first employed by Broeckmann. The sterilization should be for three hours at a temperature of 140° C. None of the twenty lots of gut thus treated was found to contain bacteria His observations lead him to the following conclusions:

1. Catgut can be sterilized by dry heat with but slight loss of tensile strength.

2. Dry paraform gas is of doubtful value for the sterilization of catgut.

3. Methods of sterilization which involve soaking in antiseptic solutions tend to weaken the catgut to a greater or less degree.

Suturing and Wounds of the Heart.—WEHR (*Centralblatt für Chirurgie*, July 8, 1899) was led by the recent advances made in the surgery of heart wounds to study experimentally on animals the technique of such operations. His studies lead him to the following conclusions:

1. The heart must be reached through a large wound if the operation is to be successful on a wounded and bleeding heart. He found it very difficult to operate unless there was given sufficient room; the sutures could not be placed with ease otherwise or with the rapidity that the urgency of the case demands.

2. Some method is essential by which the heart can be freely exposed and the wound enlarged in any needed direction.

3. Compression of the heart to make the operation bloodless is dangerous. He found that in the dog it produced death, and believes it should be strenuously avoided in the human being.

The author proposes the following method for operations on the heart:

An incision commences on the right border of the sternum, just above the fourth rib, and passes transversely across to above the cartilage of the fourth rib on the left side. The incision is then curved across the fourth, fifth, and sixth costal cartilages, and sweeps inward transversely through the seventh, resecting the xiphoid at its base. The costal cartilages are to be resected in an inclined direction, so that the plane of the incision intersects the cartilage nearer to the sternum internally than it does externally. After the cartilages have been resected and the sternum has been sawed through without injury to the mediastinal periosteum the whole skin muscle and bone flap are turned back gradually, while the costal pleura is carefully dissected free from it. When this is accomplished the flap is turned clear back, breaking the costal cartilages on the right side. In resecting the ribs a button-pointed bistoury should be used, to avoid injury to the internal mammary artery.

Excision of Carcinoma of the Sigmoid Flexure.—BARLING (*Birmingham Medical Review*, March, 1899) reports two cases of carcinoma of the sigmoid which came to him sufficiently early in the course of the disease to justify an attempt at excision. Three other cases suffered from the disease in the same location, but local and general conditions absolutely forbade any attempt at excision.

In neither case was any attempt made at the time to restore the continuity of the bowel. It was undesirable in the first case, owing to the adhesion of the growth to the parietes and the rather extensive manipulation called for in the excision. He, therefore, employed Paul's method. In the second case the patient was in the middle of an acute attack of obstruction and six months pregnant. The complete operation, with restoration of the bowel, is the more brilliant operation; but the author sought the rapid recovery of his patients, and in consequence did the shorter operation. The situation of the growths made the condition of the patient after operation the same as in left inguinal colotomy. The results were satisfactory in these two cases, the pregnant woman recovering and giving birth to a healthy child at term.

They were, however, disappointing, in that recurrence took place at the end of twelve months, though the conditions at the time of operation were apparently very good. The cases were not seen sufficiently early to render the chances favorable.

The Surgical Sequelæ of Influenza.—FRANKE (*Archiv f. klin. Chirurgie*, 1899, Band lix. Heft 1 and 3) finds that the involvement of bones is not an infrequent sequela of influenza. The manifestations are very similar to those seen after typhoid fever. They are chiefly osteoperiostitic in form, with fewer cases of osteomyelitis. This is the great difference from most forms of infectious bone disease, as the majority of them are forms of osteomyelitis. Suppuration takes place in a very small proportion of cases. In his own cases necrosis never took place, or at least not to the formation of sequestra. In some cases there was no suppuration, but the bone was softened. As an illustration, he mentions a case in which softening followed a traumatism in a patient who had had a severe attack of influenza.

The causation of all these bone diseases is possibly not proved, but it is certain that the influenza bacillus or the toxins it produces are responsible for some of them. This infection is less severe than that produced by the staphylococcus or streptococcus, and is more prolonged in its development. The bacillus can apparently remain dormant for a considerable period before manifesting its presence in these sequelæ.

Operation for Perforation in a Case of Typhoid Fever.—MARTIN (*University Medical Magazine*, June, 1899), in reporting a case in which he operated for perforation, but in which the patient died sixteen hours after operation, says of these cases that the operation will become more frequent, but that the mistakes in diagnosis and operations where perforation has not taken place will tend to give a set-back to the legitimate operation. There are certainly cases in which the differential diagnosis is extremely difficult. In intestinal paresis there is liable to be an absence of pain or fixed tenderness, and of the hardness of the abdomen which is a common accompaniment in peritoneal inflammation. Moreover, shock and a falling temperature are not characteristic of this condition. When, however, a patient in the second or third week of typhoid fever complains of a sharp, sudden, persistent, agonizing pain; of a great increase of tenderness; when this is accompanied by symptoms of marked constitutional depression, and is followed by rapid and life-threatening tympany, if there is the slightest prospect of his surviving operation the abdomen should be opened at once.

Deformity and Loss of Function Following Colles' Fracture that Involved the Radial Epiphysis, Retarding its Growth, while the Ulna Continued to Grow.—MARCHANT (*Rev. d'Orthopedie*, July, 1899) reports the case of a youth, aged eighteen years, who had fallen from a tree at the age of nine, fracturing both wrists. They had been set, and united well, with no loss of function. For some time previous to presenting himself the patient had noted an increasing functional loss, and finally observed a marked deformity of the left wrist, which brought him to the hospital. The examination showed a prominence of the ulna that was very marked. A skiagraph demonstrated the correctness of the opinion that there had been an interference with the development of the radius, due to the fracture which had involved the epiphysis, while the radius had continued to grow. A resection of the ulna corrected the deformity, and as a result perfect function was secured in the joints.

The Arterial System of the Kidney and its Relation to its Pathology and Surgery.—ZONDEK (*Archiv f. klin. Chirurgie*, 1899, Band lix. Heft 3) has made a careful study of the arteries of the kidney and their distribution by means of corrosive preparations, both in normal and anomalous cases. From these studies he derives the following conclusions that have a direct bearing on the surgery and pathology of the kidney:

1. The fact that one pole of the kidney frequently receives its arterial supply from a distinct branch of the renal artery would seem to account for tubercular infection of the kidney often being unipolar.

2. The origin of the capsular arteries is from the renal arteries within the parenchyma. This explains the frequent combination of paranephritis in the foci located in the substance of the kidney. The virus that should pass out through the kidney can enter the kidney and the capsule as well. Thus in paranephritis when the kidney is exposed disease is frequently found in its substance which was the original pus and yet had produced no clinical symptoms, or the reverse may be true.

3. The course of the capsular arteries dividing between the anterior and posterior surfaces and the superior and inferior poles of the kidney explain the division of paranephritic abscesses and processes into four groups, as pointed out by Tuffier.

4. The fact that occasionally we find a second anomalous main branch of the renal artery outside of the hilum, which is distributed directly to one or the other pole, accounts for the fact that occasionally during nephrectomies hemorrhage persists after the arterial pedicle has been ligated.

5. The relations of the different branches of the renal artery to the different portions of the kidney explain the mode of infection which produces localized metastatic abscesses.

6. The resection of the kidney is possible, as has been shown by various operators, and is indicated under certain conditions, as the function of the kidney can be maintained in the remaining portions. The distribution of the arteries indicates the lines of incision if the functional activity of the kidney is to be maintained at its best. The diseased foci should be removed in wedge-shaped sections, having the base on the periphery, with the lines of incision made in reference to the arterial distribution.

7. In nephrotomies the median incision should not be employed, but the arterial distribution should be taken into account. The section should be made in the middle third of the lateral aspect, parallel to but one-half to three-quarters of a centimetre from the middle line on the dorsal aspect of the kidney. It should penetrate somewhat obliquely into the pelvis. This incision avoids the larger arteries, and is less liable to provoke serious hemorrhage.

The Resection of the Vertebral Arches in Cases of Paresis due to Spondylitis.—TRENDELENBURG (*Archiv f. klin. Chirurgie*, 1899, Band lix. Heft 3) derives the following conclusions from his observations:

1. In pressure-palsies due to spondylitis the pressure upon the spinal cord is exerted either by an abscess or caseous focus in the spinal canal, by pachymeningitic granulations and fungous growths, or by the narrowing of the canal as the result of the angular deformity. The latter is far more frequent than has been formerly supposed.

2. The resection of the vertebral arches in selected cases has a more favorable prognosis than was known previously. It is especially indicated in cases that are nearly or completely cured of their tendency to angulation, while in acute cases the prognosis is bad, and operation may make the condition worse.

3. Complete paralysis does not preclude benefit. Even in cases that have existed seventeen years there may be and has been relief.

4. The result of the operation, as in other cases of nerve operation for pressure-palsies in the extremities or neuroplasties, can be delayed for a con-

siderable time. It is generally a year or six months before the paresis has completely disappeared.

The Operative Treatment of Epilepsy.—SCHAR (*Archiv f. klin. Chirurgie*, 1899, Band lix. Heft 3) comes to the following conclusions as the result of his study of the literature of this subject and his observation of the cases occurring in Koch's clinic:

Operation is to be undertaken in cases of epilepsy whenever internal dietetic treatment has failed to produce results in a short time. The slight mortality from such operations makes the danger inconsiderable. It is better to operate too often than too seldom. The surgeon who treats a case of traumatic epilepsy by internal medication for a long period, and only resorts to operation when he finds he cannot produce a cure by other means, does not do the best for the patient. Epileptic patients should be examined by the surgeon as well as the physician at an early period. It is best for them to make the examination together. In asylums the surgeon should from time to time examine the cases and determine which require immediate operation.

The surgeon should not forget, however, that internal medication and dietetic treatment are of great value, and that without them, even after operation, the patient could not recover. Alcohol must be avoided and special care given to the diet of children who have suffered from traumatism. Epilepsy due to traumatism can be greatly benefited by operation at the site of the trauma.

The Treatment of Compound and Complicated Fractures of the Leg.—CHANDOYE (*Rev. d'Orthopedie*, July, 1899) arrives at the following conclusions regarding the method of treatment employed by Poucet:

1. The treatment of fractures of the leg situated in its diaphysis and compound in character varies with the severity of the injury.

2. In the less grave cases, where there is a small wound but no bone protruding, after strict antiseptic precautions have been employed, the limb can be placed in proper position an ordinary fixation applied, together with an antiseptic dressing.

3. In the next group of cases, where the bone protrudes from a wound of greater or less extent, the result of a severe traumatism, recourse should be had immediately to resection, with careful antisepsis. This operation should be done immediately, before general infection has taken place. No more should be removed than is absolutely essential. The wound should not be enlarged or more dissection done than is absolutely necessary. It should, however, be sufficient to permit perfect antisepsis and the reduction of the fragments into position.

4. Should the case be seen later, after the infection has become more general, excision reduction, with rigorous antisepsis, is still the best procedure.

5. Amputation should be avoided in the great majority of cases, and only employed where the bloodvessels or nerves have been so injured that the vitality and functional value of the limb cannot be depended upon. Secondary amputation is to be employed only when a persistent infection and the weakened condition of the patient make it absolutely necessary.

6. The remote results of resection, so far as the functional ability of the limb is concerned, are excellent. The author has collected fifteen cases, which were seen at periods varying from five to twelve years after the accident. They all employed their limbs equally with the sound one, and walked long distances without any ill effects. In some cases even as much as twelve centimetres were resected without any apparent influence on function.

PEDIATRICS.

UNDER THE CHARGE OF

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Blennorrhagic Peritonitis in Female Children.—ROUSSEAU, in his recent thesis (*Péritonite Blennorrhagique chez la Petite Fille*, Thèse de Bordeaux, 1899), discusses this most serious though rare complication of specific vulvo-vaginitis in children. Clinically, it may appear as a generalized or localized acute peritonitis of grave or benign form, or may assume a chronic type.

In the acute forms the principal symptoms are abdominal pain, spontaneous or elicited by pressure, meteorism, vomiting. The temperature is elevated, the pulse small and frequent, the countenance pinched. In the benign form the symptoms are often obscure, being those of peritonism rather than true peritonitis. The chronic variety usually gives few symptoms, and is apt to be overlooked by both patient and caretakers.

In all cases of unexplained peritonitis in girls it is important to look for a vulvo-vaginitis. The discovery of the gonococcus in the peritoneal exudate, while inconstant, is a definitive diagnostic sign.

The prognosis is grave in some of the generalized cases, but recovery may occur with obliteration of the tubes and permanent sterility.

In the treatment prophylaxis is most important. No case of vulvo-vaginitis should be considered too trivial to demand careful treatment.

If the disease seems benign medical treatment may be sufficient (leeching, laudanum cataplasms, ice poultices, opium, restriction of diet, etc.). Should the pulse become frequent, operation should at once be made by a median incision. The earlier the intervention the greater will be the chance of success.

Cerebral Complications of Pertussis.—Reference is made to a case recently reported by E. SCHREIBER (*Archiv f. Kinderheilkunde*, 1899, Band xxvi. S. 1) for the purpose of calling attention anew to the very serious cerebral complications of whooping-cough. The patient was a girl, aged two years, who,

during a paroxysm of cough, presented generalized convulsions. These recurred on the following day, and two days later were repeated, after which it was found that the child presented a paralysis of all the members, with aphasia, paresis of the pupils, right facial palsy, and relaxation of the sphincters. The temperature rose to 103° and remained elevated for two days.

Meningitis was at first diagnosticated, but the subsequent course of the case disproved this, the convulsions ceasing after twelve days, and speech and power beginning to return eight days later. The author attributes the disturbance to a meningeal hemorrhage, and concludes from a study of the literature that cerebral disturbances occurring in the course of pertussis are most often due to meningeal hemorrhage.

[In this connection attention may be called to a comprehensive study of the paralyses of whooping-cough, published last year by LEROUX (*Journal de Clinique et de Thérapeutique Infantiles*, 1898, Nos. 13 and 17), an abstract of which was made in this department of THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, April, 1899.]

Varicella Aggravated by Dermal Irritation.—P. DESOIL (*Écho Médical du Nord*, 1899, No. 17, p. 193) calls attention to the fact that the eruption of varicella may become aggravated not only by confluence of its vesicles, but also by modification of the primary lesion, which can become spontaneously pustular. In these cases the vesicle, instead of drying and becoming a crust on the third day, becomes umbilicated and is transformed to a pustule which undergoes suppuration. The temperature again rises to 102°, 103°, or 104°, and remains elevated during the whole duration of suppuration; then, if there be no other complications, the suppuration subsides about the eighth day, the temperature falls, the pustules desiccate, and finally leave indelible, white cicatrices. In brief, the disease follows the course of variola.

The author has observed two cases of this kind. The first was that of a girl, aged three years, who, on account of a bronchitis, had had the chest painted with tincture of iodine two days before the eruption appeared. The area of the back irritated by the iodine became covered with a confluent eruption, while the rest of the body showed only discrete lesions. All the vesicles upon the irritated region, without exception, became pustular and healed slowly, leaving varioliform cicatrices, while only a few upon other portions of the body followed this course.

The second case, observed during an epidemic of varicella, was in a somewhat delicate boy, aged two-and-one-half years, who had had some evidences of lymphatism. He presented the usual prodromal symptoms of varicella. The parents, fearing convulsions, put the child for ten minutes in a strong mustard bath. This energetic revulsion of the skin produced an immediate effect upon the eruption, which, from being discrete in the evening, became very confluent on the following day. Five to six hundred vesicles and bullæ of all dimensions were counted. Most of these vesicles developed into pustules and suppurated. Only a small number dried and crusted without going further. During all the period of suppuration the temperature was high and the general state bad. Finally, after local accidents (dermic and glandular abscesses) and bronchopneumonic foci, the child recovered after an illness

of six weeks, with numerous varioliform cicatrices upon the trunk and limbs, and some upon the face.

The accuracy of diagnosis was confirmed by the fact that eight months previously the child had been satisfactorily vaccinated and that at the time there was no epidemic of variola or varioloid.

An Epidemic of Infectious Catarrh due to the Pneumococcus.—M. Stooss (XXXI^e *Bericht des Jenner'schen Kinderspital in Bern*, 1898) observed during the winter of 1898, in Bern, a limited epidemic of a grippe-like disease, which, according to his studies, seemed to show the absence of the influenza bacillus. The affection began suddenly, without appreciable prodromal period, by elevation of temperature and catarrhal inflammation of the mucous membranes. This almost always localized itself in the pharynx, giving rise to an intense redness of the tonsils, pillars, uvula, and pharyngeal walls. In no case was there any formation of false membrane.

Ordinarily there was an accompanying catarrh of the mucosa of the nares, conjunctivæ, larynx, and larger bronchi. The frequency of pain in the ears showed that rarely did the Eustachian tube escape, but in no case did a true median otitis supervene. Several times there was catarrhal stomatitis and labial herpes.

Systemic disturbance was shown in all these cases by insomnia, agitation, and high fever (102°–104° F.) of remittent type. As complications, descending bronchitis or bronchopneumonia was observed. The duration of the affection, when uncomplicated, was six to seven days. All the children were under seven years of age.

Examination of the pharyngeal secretions invariably showed innumerable pneumococci, beside occasional staphylococci and streptococci. In none of the cases was the bacillus of influenza found.

Thrombosis of the Superior Longitudinal Sinus.—Haushalter (*Revue Mensuelle des Maladies de l'Enfance*, August, 1899, p. 373) reported to the Société de Médecine de Nancy, at its session, April 12, 1899, the notes of an interesting case in which this lesion was found at autopsy in an infant, aged ten months. At the age of four months it had suffered from a severe attack of gastro-enteritis. From that time till its death it had presented at intervals peculiar phenomena, characterized by crises of convulsive respiration, short cries, cyanosis, and convulsive movements of the eyeballs. These attacks lasted two to three hours and occurred several times daily. During a paroxysm the child became cyanosed to an extreme degree; it died during an attack six days after coming under observation in the hospital.

At the autopsy the only lesion found was a thrombosis of the superior longitudinal sinus, with minute thrombi disseminated in the veins of the pia mater, and intense congestion of the meninges.

The interest of the observation lies in the peculiarity and the long duration of the convulsive attacks, caused by the very slowly progressive character of the thrombotic lesion.

THERAPEUTICS.

UNDER THE CHARGE OF

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Dilatation of the Stomach; Etiology and Treatment.—DR. J. A. LICHTY discusses treatment under four heads:

1. *Hygienic.* All forms of corsets and corset-waists are condemned. Clothing is not to be suspended from the hips. Union undersuits are recommended, and a waist so constructed that when skirts are buttoned to it the weight falls on the shoulders and not on the hips. To accomplish this there are three essentials: (*a*) The material for the waist must be so cut that the pull of the skirts is in the direction of the weave of the goods; (*b*) exact fit, allowing for respiration; (*c*) buttons for skirts must be high enough to prevent pulling below the line of smallest circumference of patient's waist.

2. *Dietetic.* A rather unusual regimen is advocated—namely, two glasses of milk, with two raw eggs, at 8 A.M., 1 P.M., and 6 P.M., and two glasses of milk at 11 A.M., 4 P.M., and 9 P.M. After these meals the patient lies flat on the back or on the right side for thirty minutes; the weight of the food is thus supported by the posterior wall of the stomach instead of the greater curvature. The kidneys become active, bowels become more regular, and there is gain in weight. When the normal weight is regained a solid meal is permitted at 1 P.M., consisting of a mutton chop and zwiebach, with about four ounces of water. For the 4 o'clock feeding are substituted about twelve ounces of water. Gradual return to solid food is made. It is best to intermit the feedings of milk and raw eggs for twelve hours every ten days. During this time water is given in equal proportions in *lieu* of the milk. Foods causing fermentation must be avoided, and among these are potatoes, rice, and white bread. Raw fruits and excess of sweets and fats must also be excluded.

3. *Physical.* Abdominal massage. Exercises to develop the abdominal muscles, electricity, alternate hot and cold packs, or douches applied to the abdomen. The intragastric electrode is mentioned, but percutaneous electric stimulation is considered equally efficacious.

4. *Therapeutic.* Tincture of nux vomica (or strychnine) is very useful in atonic dilatation. The addition of quinine sulphate is often of value. When there is diminished hydrochloric acid, small, often repeated doses of the acid are beneficial following the meals of solid food. Where hyperchlorhydria exists, sodium bicarbonate, calcined magnesia, and bismuth subnitrate are useful. If constipation persists in spite of the dietetic and physical measures above mentioned, glycerin suppositories are of service. Prognosis is good if full control can be had of the patient. The symptoms will often disappear long before the stomach resumes its normal size.—*The Medical Press*, 1899, vol. lxvii., No. 3134, p. 557.

Orexin.—Dr. Eugene S. Yonge notes that the loss of appetite which complicates so many systemic diseases is frequently amenable to empirical treatment alone, and demands to be dealt with *per se*. It is a symptom which especially attracts the patient's attention, and both mind and body are depressed by its persistence. The synthetic, either as basic or its salt, the tannate, seems to be generally useful. The latter form is preferred, which is found as a yellowish-white, odorless, almost tasteless powder, insoluble in water, but soluble in diluted acids, and therefore in the gastric juice. It shortens the process of digestion by stimulating the secretion of hydrochloric acid and by increasing the motor functions of the stomach. The dose is 10 grains thrice daily before meals, preferably in a hot liquid.—*The Therapist*, 1899, No. 5, p. 120.

The Treatment of Dysentery.—Dr. D. M. M. Ross finds treatment with ipecacuanha or epsom salts not satisfactory in many cases. He recommends the examination of the water drunk by the patient, and its interdiction when infected. Rest in bed is essential. Medicinal treatment, a moderate dietary, fomentations (hot) to relieve colic, removal and prevention of fecal accumulations, using ipecac, Epsom salts or castor-oil, rectal irrigations, when bearable, of potassium permanganate or boric acid solution (strength not mentioned), followed by starch and opium enemata, to relieve tenesmus, are valuable. On the appearance of fecal dejections intestinal antiseptics are substituted for irrigation, using bismuth salicylate or subnitrate, salol, resorcin, or naphtol. If there is reason to think the ulcers are slow in healing, astringent injections are indicated. Tonics and change of air are useful later. One patient died from perforation, and autopsy showed in other fatal cases how little is needed to cause perforation. Where the disease is severe warning is given against too greatly depleting the system with ipecac or Epsom salts, and an earlier recourse to intestinal antiseptics is recommended. Opium is best not used, as a rule; sometimes it is indicated, and its action is satisfactory. In every instance the circulatory system should be carefully watched.—*The British Medical Journal*, No. 2002, p. 1151.

Acute Dysentery.—Dr. J. T. Anderson recommends for an adult three ounces of magnesium sulphate divided into three doses, and one given every hour until the whole has been taken. Aside from the depletion and thorough drainage or cleansing process, it is claimed that sulphur is set free in the alimentary canal and antisepsis is obtained. If ulceration has become established, 30-grain doses of bismuth should be given every three hours. Strychnine arseniate is an excellent general tonic. The colon should be flushed several times daily with water containing either silver nitrate or zinc sulphate.—*Medical Mirror*, 1899, No. 7, p. 337.

Malignant Œdema Cured by Injections of Carbolic Acid.—Dott. Livio Amadori reports one instance in which the œdema was seated in the left upper eyelid, later spreading over the head and closing the right eye. Seven injections of one-half grain each were made into the swelling, and on the next six one-third of a grain. This treatment was followed by a rapid fall of pulse rate and temperature, and a relief of pain and swelling, the last

completely disappearing in five days. The discoloration of the urine was not found later than thirty-six hours after the last injection. The tolerance of the patient toward the remedy is explained by the severity of the infection.—*Gazzetta degli Ospedale e delle Cliniche*, 1899, No. 67, p. 714.

The Sedative Action of Methylene-blue in Psychoses.—Dr. Pietro Bodoni reaches the following conclusions: (1) This substance has an important action upon the organism, and particularly upon the nervous system, and preponderatingly upon the elimination of the blue itself; (2) the kind of disease has a marked influence on its elimination, which varies not only with its form but also with the period of the process; (3) it is not only eliminated in the urine, but also generally in the feces of the insane; (4) the presence of chromogen and not of the blue in the urine does not indicate any changes in the kidneys as such, but a disturbance caused by other factors; elimination of chromogen takes place either from a retardation of absorption and metabolism, by which the blue-reducing agents are changed into the chromogen, or on account of increased activity of other factors; (5) a retardation of the elimination of the blue and its leuco-derivatives is to be attributed to a general disturbance of the power of absorption of the tissues which may arise from different causes, according to the physiological condition of the organism; (6) in the insane the renal functions are less dependent upon the anatomical conditions of the kidneys than upon the general disturbances which the morbid process excites; (7) methylene-blue then can even less be utilized to control the anatomical and functional conditions of the kidneys in the insane than in other diseases, or even in healthy individuals. Noting the various effects of this substance upon nerve-tissue, blood-corpuscles, and various diseases, the results of intramuscular injections of one to one and one-half grains in fourteen patients are recorded. The general conclusion is that this substance is distinctly sedative, and should be included with anylene hydrate, sulphonal, trional, tetronal, chlorolore, duboisin, hyoscyanin, bromoform, and similar remedies used in psychiatric practice.—*Klinische-therapeutische Wochenschrift*, 1899, No. 21, S. 665.

The Antiseptic and Eliminative Treatment of Typhoid Fever.—Dr. T. V. Hubbard, after a plea for a fair trial of such methods of treatment and a statement that most of the opposition comes from those who have had no experience with these methods, goes on to explain his own practice in treating enteric fever. Basing his therapy on the fact that the fever is primarily a specific local infection, he proceeds to treat the infection locally by means of purges and antiseptics. Calomel $\frac{1}{2}$ grain, guaiacol carbonate 2 grains, podophyllin $\frac{1}{20}$ grain, are given in capsule every two hours for one or two days, depending on the condition of the bowels. This is continued until four or five intestinal evacuations have resulted for two successive days. The calomel is then omitted, and half a grain of menthol is added to the other drugs. If the bowels become inactive, as they often do, on discontinuing the calomel, small doses of salts or of Hunyadi water are given in the morning. Depending on the temperature and tympanites, at least two evacuations are required daily. If the temperature remains high or rises after four or five days' treatment, calomel is again given as above for twenty-four hours;

it invariably reduces the temperature, and results in general improvement in the patient's condition. The administration of guaiacol, menthol, and podophyllin are continued throughout the disease for their antiseptic properties. *Continuous* medication *early* in the disease is the *sine qua non* of success. Occasional spongings or ablutions with cold water are advised for the good effect produced on the peripheral circulation, the shock to the general nervous system, and the subjective sensation (?) of the patient; for the fever it is unnecessary; after the first few days, antipyretics are not needed. With this method of treatment digestion is more satisfactory, and more food is recommended than these patients received under the old régime. Average duration of the disease in ninety-seven tabulated cases was fourteen and two-fifths days. Stimulants are rarely required; delirium and complications are practically unknown. Where treatment is begun within eight days of the beginning of the disease, and systematically carried out, the temperature almost invariably returns to normal at the expiration of a week or ten days. —*The Virginia Medical Semi-Monthly*, 1899, vol iv. No. 77.

The Treatment of Autotoxic Dyspnœa.—Dr. H. Huchard discusses this symptom of alimentary intoxication. This may be due to urœmia, but in general it antedates such a condition. The ready proof that this is the case is found in the aggravation of the dyspnœa when the patient is upon a meat diet; the cause is the vaso-constriction which results from this diet. When fed exclusively upon milk, both this symptom and the marked pallor disappear. Since with this symptom there are signs of arterio-sclerosis and cardiac insufficiency, potassium iodide is indicated. Should œdema be present, theobromine in large doses is necessary.—*Gazetta degli Ospedale e delle Cliniche*, 1899, No. 65, p. 694.

The Hypodermatic Injection of Silver Nitrate Over the Course of the Vagi in the Treatment of Pulmonary Consumption.—Dr. T. J. Mays, believing that the lung lesion in pulmonary consumption is not the primary morbid change, thinks a *nervous* pathology underlies a rational therapy. He therefore uses largely strychnine, electricity, phenacetin, etc. The vagi are also locally treated by massage through the overlying structures. Cough, expectoration, and sense of oppression are benefited. More far-reaching effects were sought in the introduction of nitrate of silver immediately over the course of the vagi in the neck by arousing them from their "abnormal condition." Four to seven minims of a 2½ per cent. solution of silver nitrate (Merck's) answered well, though sometimes five minims of a 5 per cent. solution were required to bring about an inflammatory reaction. The point of injection is immediately over or behind the carotid artery, half-way between the angle of the jaw and the clavicle. To avoid puncturing either the carotid or jugular vein the skin is to be elevated and the needle introduced just beneath it. Cocaine injection (2½ per cent.) should precede the above, for the nitrate of silver is otherwise very painful. The same puncture suffices for both injections, the barrel of the syringe being removed, cleaned, and filled with the silver solution, the needle remaining *in situ*. A diffuse swelling, with redness and pain, appears. The injection is to be given on the side of the neck on which the affected lung is situated, and should be re-

peated in a week or ten days, unless the first injection is followed by too much irritation. Generally it is best to limit the injections to the affected side, with an occasional one on the opposite side. Analysis of results of above treatment in forty phthisical patients is as follows: Cough and expectoration were much benefited, and likewise dyspnœa and oppression. Through diminishing the cough sleep was much improved. Appetite grew more keen, even in patients far advanced in the disease, and the general strength improved. The physical signs also exhibited a marked change for the better, so much so that it was not merely a coincidence. Gain in weight was the rule, even in the sickest patients. The writer deduces from the above that best results are obtained: in the incipient stage, both as regards symptoms and physical signs, and in an advanced stage, both are benefited, sometimes remarkably. In a great majority in a far advanced stage, cough, expectoration, and other symptoms are temporarily ameliorated, but the local lung condition is unchanged. To the above method of treatment, hygiene, dietetics, and internal medication must be added. For the purposes of study these latter were omitted in the above cited group. These injections of silver nitrate may be valuable in asthma, chronic bronchitis, and in the treatment of the prephthisical stage in the young.—*Boston Medical and Surgical Journal*, 1899, vol. cxl. No. 6, p. 133.

GYNECOLOGY.

UNDER THE CHARGE OF

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Torsion of the Uterus by Tumors.—EHRENDORFER (*Monatschrift für Geb. u. Gyn.*, Band ix. Heft 3) reports the following case: Abdominal section was performed for the removal of a sessile fibroid tumor the size of a man's head in a woman thirty-six years old, whose principal symptom had been inability to empty her bladder for several months without the aid of a catheter. On opening the abdomen the uterus was seen to the left of the neoplasm, twisted entirely around its long axis, the bladder being drawn high upward and to the left. The tumor was so impacted in the pelvis and adherent to surrounding organs that it was removed only with great difficulty, the stump being secured with an elastic cord, and treated by the old extraperitoneal method. The convalescence was satisfactory, the patient being able to pass her urine on the third day.

Supravaginal Amputation of the Uterus for Fibromyoma.—At a meeting of the Leipsic Obstetrical Society (*Ibid.*) a discussion of the relative advantages of total extirpation and supravaginal amputation developed the fact that the latter was the favorite method. ABEL thought that the size of the stump was immaterial. Only two cases of subsequent sarcomatous degeneration had been reported. In young subjects it was important, he

believed, to spare one ovary, and, if possible, a portion of the uterine mucosa, in order to preserve the function of menstruation.

SAENGER held that the possibility of malignant degeneration of the stump was so slight that it did not furnish an argument in favor of total extirpation. He was sure that a stronger pelvic floor remained after amputation.

ZWEIFEL said that for several years he had not observed exudates around the stumps. He made it a practice to leave as large a stump as possible, in order to preserve some of the mucous membrane.

Remote Results of Hysteropexy.—SEGOND (*Comptes Rendus de la Soc. d'Obst. de Gyn. et de Paed.*, April, 1899) reports a case of hysterectomy in a case in which he had performed hysteropexy six years before. The patient was relieved for two years, when she began to complain of dragging pains in the abdomen, although the uterus remained fixed to the wall. Previous to the operation she suffered from severe abdominal pain, tympanites, and evidences of intestinal obstruction. Examination showed that there was a certain range of mobility of the uterus, so that a pocket existed between the fundus and the anterior wall of the abdomen, in which it was inferred that a coil of intestine might have become caught. As the patient was near the menopause, the writer decided to perform hysterectomy. On opening the abdomen the fundus uteri was found to be attached to the parietal peritoneum by eight small bands, each about an inch in length, corresponding in number to the silk sutures which had been used; no trace of these sutures remained. Although no adherent intestine was seen, it was apparent that the conditions were favorable to imprisonment of a loop of gut in the space left in front of the uterus. The patient made a perfect recovery and remained well.

[This case furnishes a strong argument for the abandonment of ventrofixation and suspension in favor of internal shortening of the round ligaments. —ED.]

Operations on the Cervix Uteri from an Obstetrical Stand-point.—In a discussion on this important subject before the Paris Obstetrical Society (*Ibid.*), following the reading of a communication from Pinard, BOUILLY calls attention to the fact that if trachelorrhaphy or excision of the cervix is properly performed there is less likely to be dystocia than in cases in which the diseased tissue is not thoroughly removed.

DE RIBES had seen only one case in which delivery was rendered difficult by previous amputation of the cervix, as the tissues usually softened under the influence of pregnancy. In this instance manual dilatation overcame the obstruction.

SEGOND admitted that cases might occur in which rigidity of the cervix during labor would result as the result of excision, even when the operation was properly performed. He therefore believed that it should be limited to cases in which it was clearly indicated, and should not be performed indiscriminately where a simple curettement was often sufficient.

PORAK thought that gynecologists were not in a position to present reliable statistics, since only a small proportion of the cases were kept under observation. He cited two cases, in one of which he was called to perform em-

bryotomy upon a woman who was moribund after being in labor for several days. The cervix, which had been amputated, remained rigid and undilatable, so that dystocia was complete. In another instance labor had continued for five days, on account of rigidity of the cervix, resulting from previous amputation, and rupture of the uterus occurred, which was treated successfully by immediate cœliotomy and suture of the tear. In the writer's opinion rigidity of the cervix in these cases was due to incomplete removal of all the cicatricial tissue during the operation.

LEPAGE was of the opinion that non-dilatability of the cervix during labor was due not only to failure to obtain primary union after excision, but to the fact that too radical an operation was often performed, so that no vaginal portion whatever remained. It was easy to understand that in the event of a laceration of such a cervix the lower uterine segment would be apt to be involved. In short, it was important for the surgeon to bear in mind when operating upon the cervix not to remove any more tissue than was necessary, in order not to interfere with the physiological development of the gravid uterus.

PINARD, in closing the discussion, stated that rigidity of the cervix during labor was not confined to operations performed for the removal of diseased tissue, and cited two cases of simple incision for stenosis in which labor was attended with the same complication which had been described. The speaker was in favor of immediate trachelorrhaphy in cases of extensive laceration, by which subsequent cicatrization was prevented.

The Germ Theory as Applied to Gynecology.—In a recent address before the British Gynecological Society, BANTOCK (*British Gynecological Journal*, May, 1899) provoked a vigorous discussion by declaring his opinion that the presence of specific micro-organisms was "the result and not the cause of disease." He stated that he was "still content with making his hands as clean as an ordinary washing with soap and water would make them, thus removing Lister's 'grosser forms of septic mischief.'"

It is hardly necessary to state that the consensus of opinion was entirely opposed to the eminent reader, whose paper was regarded as an individual confession of faith rather than a scientific argument.

Diffuse Adenoma of the Endometrium.—OLIVER (*Ibid.*) reported the case of a virgin, aged thirty-four years, who had menstruated profusely for ten years. On examination a firm uterine tumor was felt, extending two and a half inches above the symphysis pubis. Two mucous polypi protruding from the cervix were removed, and the hemorrhages were checked for two years, when they returned and became continuous. The polypus was excised and a teacupful of soft tissue was removed from the uterine cavity, which presented the microscopical appearance of simple adenoma. The uterus was subsequently reduced so that it could not be palpated in the abdomen, but two months later the patient returned complaining of a constant watery discharge. A second curettement resulted in the removal of another cupful of adenomatous material, but the watery discharge continued, so that abdominal hysterectomy was performed successfully five months later.

The uterus weighed nearly two pounds, and, incising it, the entire endometrium was seen to be studded with nodules varying in size from a pea to a walnut, a few being polypoid. There was general thickening of the muscular wall, with general evidence of infiltration of the neoplasm.

The reporter hesitated about regarding the condition as malignant, on account of the history of the case, which showed that the growth had probably existed for several years. The patient was the youngest in whom diffuse adenoma of the endometrium had been reported.

Vapo-Cauterization of the Uterus.—VAN DE VELDE (*Centralblatt für Gynäkologie*, 1898, No. 52) reports a fatal case in which steam at 105° C. was introduced into the uterus for five minutes for the relief of climacteric hemorrhages. The patient died of septic peritonitis on the fourth day after the operation. Autopsy showed that a perforation had occurred at the fundus. The writer adds a warning against the use of vapo-cauterization for hemorrhage in which there is the risk not only of perforation, but of subsequent obliteration of the uterine cavity and complete atrophy of the organ. He would limit its application to the treatment of inoperable carcinoma.

PINCUS (*Centralblatt für Gynäkologie*, 1899, No. 4) criticises the foregoing report, affirming that the accident was due not to his method but to its faulty application, since the introduction of the steam should be limited to half a minute. He claims that by adhering strictly to the rules laid down by him the accidents and subsequent ill results referred to cannot occur.

OBSTETRICS.

UNDER THE CHARGE OF

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Three Cæsarean Sections upon the Same Patient.—In the *Centralblatt für Gynäkologie*, 1899, No. 15, POLLAK contributes a very interesting paper upon this subject. He has collected thirty cases, in which two Cæsarean operations were performed upon the same patient. He has found twelve cases in which the operation was performed three times, four cases in which four children were delivered by this method, and one case in which a mother bore five children by repeated Cæsarean section. His own case of three operations upon the same patient was as follows:

When the patient first came under observation she was nineteen years old. She had a rhachitic pelvis, and labor was induced and terminated by version, followed by craniotomy.

When her next pregnancy occurred the usual conservative Cæsarean operation was performed, the womb being closed by buried silk sutures, and the

peritoneum united by fine continuous silk. Mother and child made an excellent recovery. The second operation was more than a year later, and was successfully performed in the same manner. There were adhesions between the mesentery, the uterus, and the abdominal wall, but these were very readily separated. Mother and child made a good recovery without complications. The children were rhachitic, and shortly after the second pregnancy both of them died. When the patient again became pregnant she declined any form of delivery except the Cæsarean operation, because she desired a living child. This was accordingly performed in the same manner as the preceding, and with equal success.

At each operation after the first the tissues were very carefully inspected, to observe the scar which was present. The wall of the uterus was firm and solid, not thinned or abnormal. The adhesions which existed between the wall of the abdomen, the mesentery, and the uterus were not resisting, and were readily separated. Each time the incision was made so far as possible through fresh tissue, and in each operation silk was used to close the parts. Certainly nothing could have been more successful than these three operations.

Pollak calls attention to a phenomenon which has been observed by other Cæsarean operators—namely, that the child is slightly asphyxiated and delays respiration for a few moments. It is, at first sight, not easy to account for this, as the child has had but little birth-pressure. Pollak thinks it due to the anæsthetic which the mother has taken during the operation.

Complete Rupture of the Uterus.—At a recent meeting of the Gynecological Society of Dresden, LEOPOLD (*Centralblatt für Gynäkologie*, 1899, No. 15) reported the case of a woman aged thirty-five years, who had borne four children, and who was in labor with the fifth. The left shoulder presented, and the progress of labor ceased. On admission to the clinic the patient was found considerably shocked, and rupture of the uterus was diagnosticated. She was immediately prepared for operation and the abdomen was opened. Considerable clotted and fluid blood was found in the abdominal cavity. The child was lying across the abdomen, its head covered by the membranes. After the child and appendages had been removed a transverse laceration across the anterior wall of the uterus was found. This extended through the broad ligament of the right side, and the serous membrane had been separated for some extent by blood clot.

As it was impossible to suture the uterus, it was amputated, gauze drains were inserted, and the abdomen was closed. Death followed on the ninth day afterward, and post-mortem examination showed purulent peritonitis, with partial occlusion of a portion of the small intestine. Upon investigating the history of the case, a midwife had been in attendance, who had failed to promptly summon the aid of a physician. She was accordingly fined for this neglect.

A Case of Rupture of the Uterus Treated by Vaginal Extirpation.—In the *Centralblatt für Gynäkologie*, 1899, No. 13, SOLOWIJ contributes the interesting report of a case of ruptured uterus, in which the womb was removed by vaginal extirpation.

The patient was thirty-four years of age, and had borne two children spontaneously. When admitted to the hospital the abdomen was distended and painful. The fœtal heart-sounds could not be heard, and a bloody discharge was present. The placenta could be felt in the vagina, but the external os could not be reached. On introducing the hand to perform version the operator discovered that the anterior wall of the womb was torn above the cervix. The fœtus was in transverse position, between the uterus and the abdominal wall.

After the patient had been anæsthetized the placenta was first removed, and the right foot of the child brought down. The child was delivered without great difficulty and its appendages removed through the uterus. There was no hemorrhage, and the womb was readily removed through the vagina. The patient, however, perished in shock within a few hours.

When a post-mortem examination was made it was found that the tears in the uterus were extensive, and that a hæmatoma had formed under the serous membrane.

The Use of the Rontgen Rays in Diagnosticating Pelvic Deformities.—ALBERT recently demonstrated to the Gynecological Society of Dresden (*Centralblatt für Gynäkologie*, 1899, No. 15) his method of obtaining a tracing of the brim of the pelvis and the position of the child by the use of the Röntgen rays. He obtained the best results in examining the superior plane of the pelvis, and was successful in diagnosticating flattened pelvis. In one of his cases the patient laid flat upon the back, and in others assumed a partially sitting posture. He was enabled by these means to obtain a measurement of the upper portion of the pelvis, which gave a good indication of its capacity. He exhibited diagrams illustrating these researches, and called attention to the difficulties arising from the fact that the shadow of the promontory of the sacrum became confused with that of the symphysis; but from three to four minutes' exposure to the action of the rays was required. He also exhibited pictures of the fœtus in the fifth, sixth, eighth, and tenth months of development.

Torsion of the Pedicle of a Dermoid Cyst Occurring After Abortion.—In the *Centralblatt für Gynäkologie*, 1899, No. 13, UNTERBERGER adds to the literature of ovarian tumors an interesting and uncommon case.

The patient was admitted to the hospital with an incomplete abortion. Curetting was advised, and on examination a dermoid cyst, very mobile, was found in the abdomen. The presence of the tumor had been discovered three years before after a confinement. The curetting was followed by no disturbance whatever, and on the fifth day afterward a bimanual examination was made. When this was done the tumor was found above and to the right side at the border of the ribs, and not so freely movable as before. This was thought to have been caused from a lack of free bowel movements. Soon after the examination the patient was taken with colicky pains, the abdomen became distended, very sensitive, and the pulse rapid, and the tumor was discovered in the left half of the pelvis. Torsion of the pedicle was diagnosticated, and the patient was operated upon as soon as possible. The diagnosis was correct. There were two twists in the pedicle to the left, with

beginning engorgement and the formation of a blood clot. The operation was fortunately successful, the patient making a complete recovery.

As regards the frequency of this accident, Küstner thinks that it occurs in 47 per cent. of dermoid cysts, Thorn in 35 per cent., Schauta in 20 per cent., Demakis in 17 per cent., while the older writers did not think it oftener than from 6 to 10 per cent. In 500 ovariotomies Spencer Wells saw this complication 12 times. The writer in 41 ovariotomies has seen 3 cases of twisted pedicle.

Subcutaneous Emphysema Complicating Labor.—Among the rarer complications of active labor is the occurrence of diffuse subcutaneous emphysema. This must evidently result from violent expulsive efforts on the part of the mother, and if accompanied by infection may be of a very serious nature.

In the *Centralblatt für Gynäkologie*, 1899, No. 15, is an account of such a case, given by MAISS before the Gynecological Society of Dresden.

The patient was a young woman, aged seventeen years, who was in active labor when she entered the hospital. The occiput was upon the right side, the head large and hard and movable above the pelvic brim. The pelvis was a symmetrically contracted and also flattened pelvis. In view of the conditions present the membranes were retained as long as possible, and as the birth-canal was small and resisting, a colpeurynter was introduced. The patient had very strong pains, the occiput rotated posteriorly upon the right side, and the patient was placed in Walcher's position, to favor the descent of the head. Labor proceeded steadily, but slowly, the heart-sounds of the child remaining good. When the head had reached the pelvic floor, after some especially strong pains, the woman's face was observed to become dusky and greatly swollen, this condition extending downward upon the chest. Subcutaneous emphysema was evidently present. The child was at once delivered, and survived its birth three hours, when it perished from hemorrhages into the medulla and beneath the dura mater.

The emphysema did not increase nor did it cause the mother especial annoyance. Cold compresses were placed upon the skin, and the swelling steadily subsided. A small incision was made in the regions of the clavicles, and thirteen days after the birth of the child the emphysema had disappeared. The mother's recovery was complicated by a foul vaginal discharge, which contained gonococci and staphylococci. She ultimately made a good recovery.

In explanation of this condition it has been alleged that during excessive labor pains, while making straining efforts, air entered the subcutaneous spaces at the apices of the lungs through rupture of a small vesicle, or gained access to the circulation in some manner through the uterus. The exact mechanism of this phenomenon is not, however, known.

Delivery of the Arms in Breech Cases.—In the *British Medical Journal*, June 10, 1899, CALLENDER describes an expedient successfully employed in a breech case, where the arms were displaced upward. He knotted a corner of a soft handkerchief and passed it around the shoulder which lay anteriorly. It was then pushed along the arm to the elbow, and kept there,

while with the other hand traction was made obliquely across the anterior aspect of the child's body. The arm was readily brought down in this manner.

Experiments Upon the Closure of the Fallopian Tubes.—In the *Archiv für Gynäkologie*, 1899, Band lviii. Heft 2, FRAENKEL gives the results of experiments conducted with a view of determining an efficient method for closing the lumen of the Fallopian tubes.

He ligated the tubes at various points with catgut, silk of different sizes, and on some severed the tubes with the thermo-cautery. Most of these attempts, however, were unsuccessful, and the lumen of the tube was found to persist, and to be reformed in spite of ligation. Various changes in the tubes followed ligation, the ligatures when silk remaining unchanged. He then resected a portion of the tube and examined the animal ninety days after the experiment. It was found that the lumen of the tube had been apparently restored, and the same result followed with the use of the thermo-cautery. In some cases hydrosalpinx formed and, in a few, adhesions.

He has collected a number of clinical cases where a considerable portion of the tube has been removed, but where pregnancy subsequently occurred, and this result is illustrated in his own experiments when he found the tube restored after amputation and ligation. He reaches the conclusion that the function of the tube can be destroyed only when the tube is entirely removed and its uterine termination exsected and the tissues closed with a flap of peritoneum.

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES.

UNDER THE CHARGE OF

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Exophthalmic Goitre.—JONNESCO (*Centralbl. f. Chir.*, February 11, 1899; *Medical News*, April 22, 1899) reports that in ten cases of true exophthalmic goitre a cure was obtained in six and improvement in four by resection of the cervical sympathetic. The exophthalmos disappeared first, then the nervousness and tachycardia, and finally the goitre. The results upon cases of secondary exophthalmic goitre were less successful.

Operations for Stricture of the Trachea.—DRS. W. S. JONES and W. W. KEEN, of Philadelphia, report (*Journal of the American Medical Association*, June 10, 1899) two cases successfully operated upon by Professor Keen. One was for removal of a diaphragm following an attempt at suicide, and completely closing the trachea above the fistula occupied by the tracheotomy-tube. Dr. Keen got access to the diaphragm by an external incision in the middle line, which led him directly upon the œsophagus, showing that the

severed tracheal rings had become separated by retraction. The diaphragm was dissected out and the two ends of the trachea sutured together. The result has been completely successful, and the patient breathes without his canula. The tracheotomy-tube was permanently removed upon the sixth day, and the fistula became closed entirely at the end of the sixth week.

We believe this to be the only case of the kind upon record.

The second case was a stricture due to a membranous diaphragm with a very small orifice a short distance below the vocal bands. In this case Dr. Keen likewise got access to the obstruction by external incision. It was found necessary not only to split the trachea for six or seven rings, but to split the cricoid cartilage also. The upper border of the stricture was found at the third tracheal cartilage, and extended over the third and fourth and to the upper border of the fifth. The fibrous stricture was dissected out and the trachea was completely closed by catgut stitches passing through the perichondrium and pre-tracheal tissues.

These operations were performed with the patients in the Trendelenburg posture.

Foreign Body in the Bronchus.—The foreign body was the tracheal portion of a hard-rubber tracheotomy-tube, which had become detached from its shield.

Dr. A. Coolidge, Jr., of Boston, by the aid of reflected sunlight, saw the foreign body in the right bronchus, its upper end about one-half inch below the bifurcation, whence it was seized by a long pair of alligator forceps and removed without difficulty. The tracheal opening had been enlarged and the urethroscope introduced to facilitate manipulation.—*Transactions of the American Laryngological Association*, *Medical News*, July 1, 1899.

Bronchocele.—Dr. Edmund W. Holmes, Demonstrator of Anatomy in the University of Pennsylvania, favors a lateral route behind the sterno-cleidomastoid muscle for gaining access to all deep-seated growths below the level of the upper margin of the thyroid cartilage (*International Clinics*, vol. ii., ninth series). He reports a successful operation for bronchocele and two for cervical sarcoma.

Stenosis of the Larynx.—In recurring laryngeal stenosis after intubation, Dr. Louis Fischer (*Journal of the American Medical Association*, May 13, 1899) indorses the opinion of Dr. O'Dwyer, that this condition was usually the result of traumatic injury, either from improperly constructed or illy adapted tubes, or from the accumulation of calcareous deposits upon the tube. He had employed with much success in these cases a method of intra-laryngeal treatment of the ulcerated surface, which, too, had been suggested by O'Dwyer, and which consisted in the use of tubes coated with medicated gelatin. The best results were obtained by immersing a rubber tube in a hot solution of gelatin containing either 25 per cent. of alum, 20 per cent. of ichthyol, or paraffin medicated with 3 per cent. of iodoform or europhen.

Complete Bilateral Recurrent Paralysis due to Carcinoma of the Œsophagus.—Dr J. Herzfeld reported this case before the Berlin Society

of Laryngology, July 1, 1898 (*Annales des Maladies de l'Oreille, du Larynx, etc.*, August, 1899).

A man, aged sixty-four years, began to experience difficulty in swallowing three years before, and, the trouble increasing, frequent dilatations with sounds were necessary. Within the last three months gradual increasing hoarseness intervened. In probing the œsophagus the instruments of large calibre encountered an obstacle about the level of the bifurcation, while smaller sounds reached to the cardia without difficulty. The laryngoscopic image showed both vocal bands in the cadaveric position, whether during expiration or phonation. The patient died suddenly of a hemorrhage of the stomach after probing. The autopsy revealed a carcinoma of the œsophagus, extending to the posterior wall of the trachea and completely paralyzing the two recurrents.

Primitive Medullary Carcinoma of the Epiglottis.—This case was reported by DR. FLATAU (*Annales des Maladies de l'Oreille, du Larynx, etc.*, August, 1899), and when first seen was limited to the epiglottis, concealing the valleculæ and a portion of the base of the tongue. It had begun three or four months after the existence of a sensation of a foreign body, difficulty in swallowing, and occasional slight pains. The growth was removed by Professor Gluck, who performed subhyoid-pharyngotomy, preceded by preliminary tracheotomy. The base of the tongue was found sufficiently involved to make it necessary to extract a portion of the hyoid bone. Fourteen days later the patient was taken with intestinal hemorrhages, the cause of which remained undetected.

Adenocarcinoma of the Nose.—At the recent meeting of the American Laryngological Association, DR. JAMES E. NEWCOMB, of New York (*Medical News*, July 1, 1899) reported a case in a female subject, aged sixty-one years, in which reproduction of tissue had followed intranasal removal. Recurrence had twice followed, in which a radical operation seemed necessary. Six other cases were mentioned to be added to those enumerated in a table prepared by Dr. Hopkins.

Suppuration of the Accessory Sinuses of the Nose.—By far the most valuable practical contribution to this subject which has been published of late is an exquisitely illustrated article by DR. JACOB E. SCHADLE, of St. Paul, Minn. (*St. Paul Medical Journal*, January and February, 1899), on the etiology and diagnosis of empyema of the accessory sinuses of the nose.

Eight photographic plates from the wet-preparation admirably illustrate the subject, the text of which will hardly bear condensation in the few paragraphs we could devote to it, so that we must refer our readers to the original; but we may say that great stress is laid upon the proper appreciation of the normal and abnormal anatomy of the parts before disease as well as after it.

Nasal Epilepsy.—E. MEYER (*Annales des Maladies de l'Oreille, du Larynx, etc.*, August, 1899) reports this case in a man, thirty-two years of age, who had been affected since he was sixteen years old. The paroxysms occurred at irregular intervals of from three to ten weeks, and each paroxysm was

preceded by an abundant purulent discharge from the left nasal passage. An empyema of the maxillary sinus was recognized, which was discharged by trephining the anterior wall and followed by the ordinary after-treatment. Since this time the epilepsy has not reappeared, but Dr. Meyer was not sure a cure had been definitive.

Fibroma of the Nasal Orifice.—This unusual case, reported by DR. ALEXANDER (*Annales des Maladies de l'Oreille, du Larynx, etc.*, August, 1899), occurred in a female, fourteen years of age. It occupied the left wing of the nose and nearly filled one-half of the nasal passage.

Neurasthenia in Certain Affections of the Nose and Throat.—DR. MARCEL NATIER (reprint from *La Parole*, 1899) reports two cases—one due to an elongation of the uvula and disappearing after section of the same, and another caused and kept up by an artificial ulceration on the nasal septum of the right side. Both occurred in female subjects.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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The Fate of Embryonic Cells Implanted in the Bodies of Adult Animals.—BIRCH-HIRSCHFELD and GARTEN (*Beiträge zur path. Anat. und allg. Path.*, Band xxvi. No. 1, p. 132).

These experiments consisted in the introduction of embryonic cells into an adult animal of the same species, but differed somewhat from previous attempts. Very young embryos were used, as their proliferation capacity is much greater. Undifferentiated cells were used to see if there would occur a later differentiation. No whole embryos or even whole limbs were used, but teased cells, that they might be better nourished and that different tissues might be reproduced. For implantation a tissue was sought of which the nourishment would be abundant, and which could easily be differentiated from the implanted tissue. For these reasons the liver was selected.

The embryos were teased out in salt solution, which was then injected under antiseptic conditions into the portal vein. Death followed after some days. Then another method was tried. After laparotomy the solution was repeatedly injected directly into the liver. Thus the exact place was noted, and later could be easily excised. By careful technique the same animal was sometimes operated on three or four times. Rabbits and their embryos were used first, then chickens, goats, salamanders, and frogs. Positive results were obtained in more than one-fourth of the cases.

Controls were made by injecting embryonal cells which had been killed by boiling.

The commonest result was the formation in the liver of cartilaginous nodules up to the size of a pea. Embryos of the same size and age as those injected showed no cartilage whatever, therefore a differentiation after implantation is argued.

The younger the tissue injected the more positive the results. Older cells are rapidly absorbed. Calcification was sometimes found, and often showed absorption when the surrounding capsule was not very firm.

Cartilaginous nodules were found in the periphery of the lung, evidently due to embolism. They showed the same structure as the growths in the liver.

In addition to the injection of embryonic cells, an attempt was made experimentally to change the condition of the liver. After two chickens had been injected a sterile platinum wire was passed through the liver, and silver electrodes at the ends sewed to the abdominal wall. Through this wire an electric current was passed five minutes each day sufficiently strong to warm the wire. In these cases the proliferation was much further advanced. Beside cartilage there were found adenoid tissue, epithelial-like cells, and pigmented epithelium.

Giant-cells were sometimes found, which the writers believe were formed as the result of the irritation of foreign bodies (injected material). These apparently were formed from the endothelium of the capillaries. They may also arise from various fixed cells, as shown by their origin from granulation cells in the periphery of a different nodule. Owing to lack of mytosis the writers regard them as arising from the confluence of many cells.

Conclusions are drawn as follows:

The tendency to proliferation is very different in embryonic and adult cells. A functional stimulus is necessary for cell-preservation and growth.

Embryonic cells differentiate after implantation. This is a specific property of the cell, and is not dependent on the demands of the entire adult organism.

A permanent existence for the new growth has not been demonstrated by these experiments. They are not tumors, as they are not progressive. But while Cohnheim's theory is not confirmed, it is not disproved.

The positive results of the experiments have been these:

1. The injection of teased embryonal tissue into the adult liver yields a tumor-like new-growth in the liver and lung.

2. In chickens, some weeks after the implantation of one to five-days' old embryos, there has arisen in the liver adenoid tissue, epithelial-like cells, and pigment cells.

3. The best results follow when the liver is periodically stimulated.

4. The implanted cells undergo further differentiation.

5. The growth is not permanent. After weeks or months there occurs encapsulation, regressive metamorphosis, such as calcification, etc., and finally sequestration and absorption by surrounding tissue.

Chronic Fibrinous Bronchitis.—SCHMIDT (*Centralblatt f. allg. Path. u. path. Anat.*, Band x. Nos. 11 and 12. The clinical record of this case is

interesting from the point of etiology. The patient, aged fifty-two years, was suddenly chilled seventeen years ago after a fright from a burnings theatre; then followed nose-bleed and cough, with expectoration of slimy sputum, but no casts. Eight years ago there were daily chills, with coughing in the morning. At present there is expectoration of branched plugs, with a peculiar feeling in the right lung. Lying on this side would cause a "kochen" in the lung. The expectoration is without effort, and in the last four weeks has occurred daily. There is some dyspnœa, and for the last six months gravel in the urine. The patient is nervous and subsists largely on a meat diet. Eczema is present on the hands and elbows. There is a faint tendency to obesity.

The plugs found in the expectoration swell in dilute acetic and hydrochloric acids, and are completely digested in a few hours at 37° C., hence the writer concludes they are mostly fibrin. They are light yellow, elastic, soft, slippery, the size of a goose-quill, and may reach a length of 12 cm. They number ten to fifteen a day, and show blood at the small ends, but never in profusion. Some of these smaller ends are actual blood-cylinders of fused corpuscles, which the writer thinks are due to congestion, with capillary thrombosis and secondary changes of red blood-corpuscles into hyaline bodies.

Cholesterin is abundant, especially near what Schmidt calls corpora lecithinoidea, which latter resemble in appearance and chemical action corpora amylacea, but there is no demonstrable nucleus and no radiate striations. The cylinders have parallel glistening sides, open ends, and their length is in inverse proportion to their width. The wall may be thickened, the centre a mere capillary tube or entirely obliterated. The contents of these tubes are transparent, and may include erythrocytes, leucocytes, and mucous corpuscles or their nuclei.

The above-mentioned corpora lecithinoidea are also within the tubes, may be united with the wall of the latter, and at the ends may even fuse with it. Hence they have the same genesis.

The cylinders are from 5μ to 30μ (mikrons) in width, and are commonest in the finest divisions of the casts. They are surrounded by many epithelial cells, filled with fat, much detritus, cholesterin, and concentrically striated lecithin-like bodies. Both bodies and tubes give no reaction with iodine. Under ether the tubes become lax, swell up, and undergo a sudden, complete destruction, with loss of refraction. Chloroform and carbon disulphide give the same results. Acids cause no change. From these reactions and the invariable presence of blood and epithelial cells and detritus, Schmidt calls the tubes "lecithinoidschlauche" and the smaller bodies "corpora lecithinoidea." The tubes he believes to arise from a peculiar change of hyaline capillary thrombi.

From his study of the case Schmidt calls attention to the following points:

The association of chronic fibrinous bronchitis and metabolic anomalies, such as meat, diet, obesity, gravel, eczema.

Congestion of lungs and heart degeneration, with emphysema and atheromatosis, and the localization of the process in the right lung.

Microscopically noteworthy are the hyaline blood-cylinders, the corpora lecithinoidea (like the prostate bodies), soluble in ether, chloroform, and car-

bon disulphide, the lecithin-like tubes with similar reaction. The presence of fatty epithelial cells is a favorable point in prognosis. The coagula are composed mostly of fibrin.

Fragmentation of the Myocardium.—STRICKEISEN (*Ziegler's Beiträge*, Band xxvi. Heft 1, p. 105–130).

One hundred and fifty cases of death by violence were investigated with reference to fragmentation of the heart muscle-fibres.

The most frequent location was in the papillary muscles and wall of the left ventricles; then the septum and the papillary muscles and trabeculæ of the right ventricle. The auricles were never affected.

The fracture was often found near the nucleus. Cell ruptures predominated, but separation of cells occurred. The intercellular tissue was normal. Hemorhages were never observed. As for the evidence of other pathological processes, fatty degeneration was rarely found in cases of fragmentation, but pigment atrophy was a predisposing cause.

In the hundred and fifty cases fragmentation was found in 56.6 per cent. They included phthisis, acute peritonitis, pneumonia, carcinoma, typhoid, sepsis, etc. Two cases of tetanus showed marked fragmentation.

The main cause lies in the violent contraction of the heart-muscle, as fragmentation was very frequently found in death by asphyxia. Its absence in fatty degeneraton is explained by a weakened heart, which cannot contract to the point of rupture and fragmentation of fibres. Pigment atrophy, on the contrary, leads to increased frangibility of the muscle elements.

A primary weakening of the heart-muscle can be excluded by the very frequent occurrence of fragmentation in cases of accidental death.

Chemical and bacterial causes have been urged, but anatomically the fragmentation is the same, and the interstitial changes are wanting.

If it occurred long before death there would be changes in the fibres as well as interstitial changes. On the contrary, the ruptured ends match and seem normal.

As to the medico-legal question of fragmentation as a cause of sudden death, the writer answers it in the negative. Such cases usually follow great efforts or acute infectious diseases. Probably the heart weakens, vascular pressure is diminished, and asphyxia phenomena follow with fragmentation. This may explain sudden deaths at the very beginning of chloroform anæsthesia. *Fragmentation myocardii* is strongly denied as the actual cause of any sudden death.

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CONTENTS.

ORIGINAL COMMUNICATIONS.

PAGE

Appendicitis. By Maurice H. Richardson, M.D. 629
The Pathology of Paralysis Agitans, with the Clinical History and Autopsical Notes of a Typical Case without Tremor. By H. C. Gordinier, A.M., M.D. . 648
Prognosis in Heart Disease. By Beverley Robinson, M.D. 671
The Management of Surgical Injuries to the Ureters. By Beverly MacMonagle, M.D. 684
The Nature of Paramyoclonus Multiplex. By Arthur Conklin Brush, M.D. . 693
Parotitis in Old Age. Report of a Probable Case. By Henry J. Walcott, Jr., M.D. 697
A Critical Summary of the Surgical Treatment of Ptosis. By Matthias Lancton Foster, M.D. 700

REVIEWS.

Text-book of Anatomy by American Authors. Edited by Frederic Henry Gerrish, M.D. 716
Electro-hæmostasis in Operative Surgery. By Alexander J. C. Skene, M.D. . 718
Materia Medica, Pharmacy, Pharmacology, and Therapeutics. By W. Hale White, M.D., F.R.C.P. Edited by Reynold W. Wilcox, M.A., M.D., LL.D. . 719
A Manual of Organic Materia Medica, being a Guide to Materia Medica of the Vegetable and Animal Kingdoms, for the Use of Students, Druggists, Pharmacists, and Physicians. By John M. Maisch, Ph.M., Phar D. Revised by Henry C. C. Maisch, Ph.G., Ph.D. 720
Materia Medica and Therapeutics, an Introduction to the Rational Treatment of Disease. By J. Mitchell Bruce, M.D. 720

PROGRESS OF MEDICAL SCIENCE.

MEDICINE.

PAGE

Rashes after Enemata. . . . 721
A Case of Acute Suppurative Cholecystitis, with Isolation of the Bacillus Typhosus, Eighteen Years after an Attack of Typhoid Fever . . 722
Phlegmonous Periœsophagitis . . 724

SURGERY.

Lesions of the Lateral Sinus . . 725
Rhinoplasty over a Metallic Frame . 726
The Partial Subcutaneous Rupture of a Flexor Tendon of Forearm, with Formation of a Degeneration Cyst 726
Interscapulo-thoracic Amputation . 726
Removal of the Lymphatic Glands in the Inguinal Region and the Iliac and Obturator Vessels in One Operation 727

PEDIATRICS.

	PAGE		PAGE
Association of Scarlatina with Diphtheria	728	Intravenous Injection of Antidiphtheritic Serum in Grave Cases	729
Transmission of the Agglutinating Substance of the Eberth Bacillus through the Milk	729	Perichondritis of the Larynx in a Case of Scarlatina	730
		Scarlatina in Young Infants	730

THERAPEUTICS.

Chloretone	731	Belladonna in Bronchopneumonias of Children	734
The Therapeutic Value of Heroin	732	Results of the Antitoxic Treatment of Diphtheria in Prussia	734
The Open-air Sanitarium Treatment of Phthisis in the British Islands	732		
Ichthalbin for Diseases of Children	734		

GYNECOLOGY.

Metrorrhagia of Ovarian Origin	735	Gynecological Operations in the Insane	735
Ligation of the Uterine Arteries in Cancer of the Uterus	735	Ovarian Extract in Epilepsy	736
Interstitial Suppuration in the Cancerous Uterus	735	Stypticin in the Treatment of Uterine Hemorrhage	736
		Primary Cancer of the Vulva	736

OBSTETRICS.

A Case of Repeated Cæsarean Section, with Transverse Incision	737	A Study of Fœtal Development by Means of the Röntgen Ray.	738
Wounds of the Fœtal Eyes Occurring during Labor	737	Rupture of the Uterus and Hysterectomy	738

DERMATOLOGY.

The Nosological Value of Eosinophilia and the Elimination of Eosinophilous Cells by the Skin	739	bœsiform Vegetations following Eczema	740
The Cutaneous Para-tuberculosis	739	Erythromelalgia and Atrophy of the Skin	740
On Chronic Primal Dermatitis	739	The Nature of Erythema Induratum	740
Suppurative Perifolliculitis and Fram-			

PATHOLOGY AND BACTERIOLOGY.

Refractory Subcutaneous Abscesses Caused by a Fungus Possibly Related to the Sporotricha	741	Investigations of the Changes in the Peritoneal Epithelium during Inflammation	742
Epithelial Metamorphosis of Thymus	741		